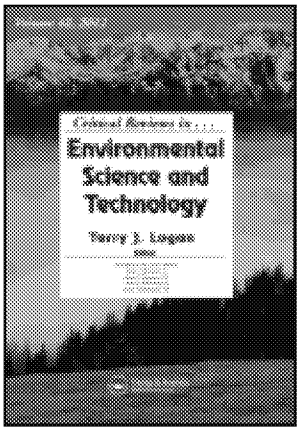

Critical Reviews in Environmental Science and Technology

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/best20

Physicochemical Properties and Aquatic Toxicity of Poly- and Perfluorinated Compounds

Guanghui Ding [a] [b] & Willie J. G. M. Peijnenburg [b] [c]

[a] College of Environmental Science and Engineering, Dalian Maritime University, Dalian, P. R. China

[b] RIVM—Laboratory for Ecological Risk Assessment, National Institute of Public Health and the Environment, Bilthoven, the Netherlands

[c] University Leiden, Faculty of Science, Institute of Environmental Sciences (CML), Leiden, the Netherlands

Accepted author version posted online: 22 Mar 2012.Version of record first published: 27 Feb 2013.

To cite this article: Guanghui Ding & Willie J. G. M. Peijnenburg (2013): Physicochemical Properties and Aquatic Toxicity of Poly- and Perfluorinated Compounds, Critical Reviews in Environmental Science and Technology, 43:6, 598-678

To link to this article: http://dx.doi.org/10.1080/10643389.2011.627016

Critical Reviews in Environmental Science and Technology, 43:598–678, 2013

ISSN: 1064-3389 print / 1547-6537 online
DOI: 10.1080/10643389.2011.627016

Physicochemical Properties and Aquatic Toxicity of Poly- and Perfluorinated Compounds

GUANGHUI DING[1,2] and WILLIE J. G. M. PEIJNENBURG[2,3]

[1]*College of Environmental Science and Engineering, Dalian Maritime University, Dalian, P. R. China*

[2]*RIVM—Laboratory for Ecological Risk Assessment, National Institute of Public Health and the Environment, Bilthoven, the Netherlands*

[3]*University Leiden, Faculty of Science, Institute of Environmental Sciences (CML), Leiden, the Netherlands*

Because of their global distribution, environmental persistence, and potential risk to human beings and ecosystems, poly- and perfluorinated compounds (PFCs) are of particular concern to scientific and regulatory communities. Despite this concern, data about the physicochemical properties and aquatic toxicity of PFCs are still limited, and there are big debates regarding the actual values of some properties investigated. In order to have a clear overview of the data available, the authors summarize the data available for the physicochemical properties and aquatic toxicity of PFCs.

KEY WORDS: aquatic toxicity, physicochemical properties, poly- and perfluorinated compounds

INTRODUCTION

Recently, organohalogen compounds have again received worldwide attention of research and regulatory communities, because of their global distribution, environmental persistence, and potential risk to human beings and the environment.[1–3] Gradually, attention has shifted from chlorinated and brominated organic compounds (e.g., PCBs, PCDD/Fs, and PBDEs to fluorinated organic compounds. Poly- and perfluorinated compounds (PFCs) are

Address correspondence to Willie Peijnenburg, Laboratory for Ecological Risk Assessment, RIVM—National Institute for Public Health and the Environment, PO Box 1, 3720 BA Bilthoven, the Netherlands. E-mail: willie.peijnenburg@rivm.nl

a class of anthropogenic fluorinated organic substances characterized by a partially or fully fluorinated alkyl chain and a terminal functional group (carboxylates, sulfonates, sulfonamides, phosphonates, alcohols). The most commonly studied PFCs are perfluorinated sulfonate acids (PFSAs) and perfluorinated carboxylic acids (PFCAs). Among them, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are of greatest concern as they are present in almost all environmental samples and are generally detected in the highest concentrations.

Given the high electronegativity and the small size of the fluorine atom, the carbon-fluorine bond possesses strong polarity and a large bond energy.[4] The strength of the carbon-fluorine bond, together with the presence of three pairs of nonbonding electrons around each fluorine atom, as well as the effective shielding of the carbon atom by the fluorine atoms render PFCs relatively stable. Consequently, PFCs are nonflammable and can resist degradation by acids, bases, oxidants, reductants, photolytic processes, microbes, and metabolic processes.[5,6] The high ionization potential and low polarizability of fluorine lead to weak inter- and intramolecular interactions that are reflected by the extremely low surface tension. This contributes to their unique hydrophobic and oleophobic nature.[7] When attached to a fluorinated alkyl chain, a charged moiety, such as carboxylic acid, sulfonic acid, or phosphate, the molecule becomes more water soluble. Then, the functionalized fluorochemical has surfactant properties because of the presence of both hydrophobic and hydrophilic moieties.[5,8]

Due to these unique properties PFCs can repel water and oil, reduce surface tension better than other surfactants, and work well under harsh conditions, and consequently PFCs have been manufactured for a large number of technical and consumer applications.[2,5,8–11] Since the 1950s, they have found wide applications including water-, soil-, and stain-resistant coatings for clothing fabrics, leather, upholstery, and carpets, and oil-resistant coatings for paper products approved for food contact, electroplating, and electronic etching bath surfactants, photographic emulsifier, aviation hydraulic fluids, fire-fighting foams, floor polishes, and insecticide formulations.[1,10,11]

Two main manufacturing processes are used to produce PFCs: electrochemical fluorination and telomerization.[5,8] Electrochemical fluorination (ECF) involves the replacement of all hydrogen atoms of a hydrocarbon by fluorine in the presence of an electric current.[9,12] During this process, fragmentation and rearrangement of the carbon skeleton can occur, producing fluorinated molecules of various carbon chain lengths and a mixture of linear, branched, and cyclic isomers. ECF was used to make perfluorooctanesulfonyl fluoride (PFOSF) based products. The majority of PFCAs and PFSAs were manufactured by the ECF process for over 50 years.[10,11,13] Telomerization involves reacting pentafluoroiodoethane with tetrafluoroethylene oligomers to yield a mixture of perfluoroalkyl iodides. These straight, even-chained, fluorotelomer iodides are then used to make a variety of telomer

products, including the fluorotelomer alcohols (FTOHs). FTOHs have been shown to be transformed in the atmosphere, soil, and metabolically in animals and microorganisms, to fluorotelomer carboxylic acids (FTCAs) and PFCAs (e.g., PFOA).[14–17]

Because of their worldwide application, PFCs have been released to the environment all over the world via various ways during manufacture, distribution, use and disposal.[18,19] There are both direct and indirect sources for PFC emissions to the environment. Direct sources result from the industry-wide manufacture and use, while indirect sources are those where PFCs are present as chemical reaction impurities or where substances may degrade to form PFCs-[10,11,20] Prevedouros et al.[10] studied the sources, fate, and transport of PFCAs in the environment. Total industry-wide global historical PFCA emissions to the environment were estimated to be 3200–7300 t during 1951–2004. These emissions are dominated by releases from fluoropolymer manufacture and comprised mainly of 8-, 9-, and 11-carbon PFCAs. Indirect sources are less important than direct sources, although there were larger uncertainties associated with the calculation of the emissions resulting from industrial sources. Paul et al.[11] estimated that the total historical worldwide production of PFOSF is about 96,000 t between 1970–2002, with an estimated global release of 45,250 t to air and water during these years. Estimates indicated that direct emissions from POSF-derived products are the major source to the environment, resulting in releases of 450–2700 t PFOS into wastewater streams, primarily through losses from stain repellent treated carpets, waterproof apparel, and aqueous fire fighting foams. Large uncertainties surround indirect sources and have not yet been estimated due to limited information on environmental degradation.

Numerous monitoring studies revealed that PFCs have been detected in nearly all environmental media and biota, reflecting their widespread global pollution.[3,6,21–24] PFCs have also been detected in human blood and tissue samples from occupationally and nonoccupationally exposed humans throughout the world.[22,23,25–27] As PFCAs and PFSAs are expected to dissociate in the environment, and have negligible vapor pressure and limited sorption to particles, they will reside in surface waters, predominantly in oceans.[10,11] The structure of many PFCs and their behavior within the body of organisms are comparable to free fatty acids (FAs), and as such they bind to liver FA–binding protein, and the protein albumin, which is mainly present in blood, liver, and eggs.[28–31]

The widespread distribution and extended residence times in the environment and organism of some PFCs have led to increased focus on their potential health risk. Recently the toxicity of PFCAs, PFSAs, and FTOHs, especially PFOA and PFOS, has been intensively studied using different kinds of laboratory rodent animals, such as rat, mouse, monkey, and rabbit. The toxicokinetic profiles of various PFCAs and PFSAs among animal models and humans have been addressed, and the biological processes that are

responsible for these toxicokinetic observations have been described. It was found that intake of PFC could lead to significant weight loss accompanied by hepatoxicity and reductions of serum cholesterol and thyroid hormones. The underlying modes of action are revealed by several commonly altered genes that involve peroxisome proliferation, fatty acid metabolism, lipid transport, cell communication, and inflammation. Various toxicity and potential modes of action of PFCAs, PFSAs, and telomere alcohols have been extensively reviewed in recent papers.[7,23,32–35]

Nowadays, monitoring studies have clearly shown the presence of PFCs worldwide, while the sources and pathways of exposure are not so clear. Basic physicochemical data are needed to clarify these aspects. However, there are limited experimental data on these basic physicochemical properties, and some controversies exist for certain properties, such as vapor pressure, acid dissociation constant (pKa), and octanol/air partition coefficient (K_{oa}). This may be related with problems in accurately assessing solubility, aggregation, sorption, and dissociation. Here, we first summarize available experimental data for the basic physicochemical properties of PFCs. This summary serves in improving the understanding of the behavior and fate of these chemicals in the environment. As PFCs prefer to reside in surface water, the toxicity of PFCs to various aquatic organisms, both freshwater and marine species, is very important for their ecological risk assessment. This aspect has also been researched and tested species include fish, invertebrates, algae, and other higher aquatic plants. To complete the overview on the fate and effects of PFCs, in another part of this article we review the available aquatic toxicity data.

PHYSICOCHEMICAL PROPERTIES

Physicochemical properties determine distributions, chemical forms, retention times, and fate of chemicals in the environment. Despite their importance, only limited research has been performed on the experimental measurement of these properties, while a lot of researches focused on environmental monitoring of PFCs. Possibly this is related to the difficulties in the experimental measurements as PFCs have unique properties unlike other organohalogen compounds, and there are only few pure chemicals. Therefore, for most of PFCs, some basic physicochemical data are still missing by now. Even for the physicochemical data that are available, there often exist controversies.

Aqueous Solubility and Critical Micelle Concentration

Since most of the PFCs have surfactant activities, they can self-assemble to form micelles above the Krafft point and the solubility in water increases

abruptly. At the Krafft Point, the solubility of a surfactant is called the critical micelle concerntration (CMC). Usually below the Krafft point the surfactant behaves as a regular organic chemical in the dissolution process, forming single molecules surrounded only by water. Above the Krafft point both monomers and micelles are observed in the water phase. The aqueous solubility of PFCs therefore has some relationships with their CMCs and Krafft points.

The CMC and the Krafft point of perfluoroalkyl acids and salts mainly depend on their chain length, chain branching, and the counterion present. It is known that in general the longer chain surfactants are more surface active and have lower CMCs than shorter chain surfactants. However, the Krafft point is elevated with increasing hydrophobic chain length. Thus, the longer chain surfactants cannot be used at room temperature. The same is true for the fluorinated surfactants. However, as fluorinated surfactants are much more surface active than their hydrocarbon counterpart, they have lower CMCs than the hydrogenated analogous with the same chain length. It has been estimated that the effect of each CF_2 group on micelle formation is approximately equivalent to 1.5 CH_2 group, which means that the CMCs of C7~C8 fluorinated surfactants are close to those of C11~C12 hydrocarbon surfactants.[36] Fluorinated surfactants in general have higher Krafft points than their hydrogenated analogue.

The Krafft points of perfluorinated carboxylates are in general lower than those of perfluorinated sulfonates, and a moderately branched chemical shows a much lower Krafft point as well as a lower melting point than a corresponding straight chain chemical. Furthermore, Shinoda et al.[36] and Kunieda and Shinoda[37] showed that the Krafft point of fluorinated surfactants is also affected by the type of counterions, but their CMCs are mainly dependent on the chain length and the valency of the counterions, while not on the type of counterions of the same valency. Divalent alkali earth metals (e.g., Mg^{2+}, Ca^{2+}) appear to yield lower CMCs than monovalent ions such as the alkali metals (e.g., Li^+, Na^+, K^+) or ammonium cations. A difference in the counterion of the same valency has less effect on the CMC. The CMCs of fluorinated acids are smaller than those of corresponding salts, which may be the result of incomplete dissociation of the acid. Fluorinated salts could be completely dissolved, while fluorinated acids will be partially dissolved, which will affect the pH values of solution, and vice versa. Therefore, the ionic concentration and pH value of solution will also have some effects on the apparent solubilities of fluorinated salts and acids.

Experimental values of aqueous solubilities are scarce for PFCs. Table 1 gives the solubilities of some PFCs. Wildlife International, Ltd. tested the solubility of the potassium salt of perfluorobutane sulfonate (PFBSK) using the shake flask method, and reported a solubility of 46.2 g L^{-1} in NANO pure water at 20 ± 0.5°C. The 3M Environmental Laboratory measured the solubility of PFBSK in water, methanol, and acetone, and found a solubility

TABLE 1. Experimentally derived aqueous solubilities (Sw, mg L^{-1}) of some PFCs

Chemical	CAS	T (K)	Sw	Method	Exp.T range	Reference	Note
PFOA	335-67-1		3.4×10^3			44	
PFBSK	29420-49-3	293.15	4.62×10^4	Shake flask	293.15	38	NANO pure water
PFBSK	29420-49-3	295.65~297.15	$5.26 \times 10^4 \sim 5.66 \times 10^4$	Shake flask	295.65~297.15	38	water
PFOSK	2795-39-3	293.15	4.98×10^2	Shake flask	293.15	40	NANO pure water
PFOSK	2795-39-3	293.15	3.26×10^2	Shake flask	293.15	40	Lab fresh water
PFOSK	2795-39-3	293.15	2.18×10	Shake flask	293.15	40	Seawater
PFOSK	2795-39-3	297.15~298.15	6.8×10^2	Shake flask	297.15~298.15	41, 42	Pure water
PFOSK	2795-39-3	295.15~296.15	1.24×10	Shake flask	295.15~296.15	41, 42	Natural seawater
PFOSK	2795-39-3	295.15~297.15	2.0×10	Shake flask	295.15~297.15	41, 42	3.5% NaCl Solution
8:2 FTUCA	70887-84-2	295.15	6.4×10	Eq batch	295.15	48	
4:2 FTOH	2043-47-2	295.65 ± 0.4	9.74×10^2	Directly measured	295.65	47	
6:2 FTOH	647-42-7	295.65 ± 0.4	1.88×10	Directly measured	295.65	47	
8:2 FTOH	678-39-7	295.45 ± 0.4	1.94×10^{-1}	Directly measured	295.65	46	
8:2 FTOH	678-39-7	295.45 ± 0.4	2.24×10^{-1}	Cosolvency approach	295.65	46	
10:2 FTOH	865-86-1	295.65 ± 0.4	1.1×10^{-2}	Cosolvency approach	295.65	47	
8:2 FTOH	678-39-7	285.15	1.34×10^{-1}	Two methods (Shake)	285~333	45	Autogenous pH
8:2 FTOH	678-39-7	298.15	1.37×10^{-1}	Two methods (Shake)	285~333	45	Autogenous pH
8:2 FTOH	678-39-7	310.15	3.18×10^{-1}	Two methods (Shake)	285~333	45	Autogenous pH
8:2 FTOH	678-39-7	333.15	2.25×10^{-1}	Two methods (Shake)	285~333	45	Autogenous pH

of 52.6~56.6 g L^{-1} in water at 22.5~24°C.[38] In an Organization for Economic Cooperation and Development (OECD) document it was quoted that PFOS has a mean solubility of 519 mg L^{-1} and 570 mg L^{-1} in pure water at 24–25°C from two studies, giving an average solubility of approximately 550 mg L^{-1} in pure water at 24–25°C.[39] In another report from the Minnesota Pollution Control Agency,[40] it was reported that solubility tests of Wildlife International, Ltd. showed that the solubilities of the potassium salt of PFOS (PFOSK) are 498 mg L^{-1} in NANO pure water, 326 mg L^{-1} in laboratory fresh water, and 21.8 mg L^{-1} in sea water at 20°C. A series of tests from 3 M on water solubility of PFOSK showed that the water solubility decreases significantly with increasing salt content. Tests showed that the solubilities of PFOSK are 680 mg L^{-1} in pure water at 24–25°C, 20.0 mg L^{-1} in a 3.5% NaCl solution at 22–24°C, and 12.4 mg L^{-1} in natural seawater at 22–23°C.[41,42] In a related study, PFOS was reported to have a mean solubility of 56.0 mg L^{-1} in pure octanol.[43] These data suggest that any PFOS discharged to a water source would tend to remain in that medium, unless it is adsorbed onto particulate matter or assimilated by organisms. The reported water solubility of PFOA is 3.4 g L^{-1}, indicating this compound is highly soluble in water.[44] Water solubility has been reported for PFOA, but it is unclear whether these values are for a microdispersion of micelles, rather than true solubility.[44] Shinoda et al.[36] measured the solubilities of several n-$C_7F_{15}COOM$ and $C_nF_{2n+1}SO_3M$ in water either by electrical conductivity (at low solubility) or by weighing the dried solution (at high solubility). However, the solubility data were shown in figures, and cannot be accurately obtained from the figures.

Kaiser et al.[45] measured the water solubility of 8:2 FTOH at 12, 25, 37, and 60°C with two modified shake flask methods. The solubility in water is 134 μg L^{-1} at 12°C, 137 μg L^{-1} at 25°C, 318 μg L^{-1} at 37°C, and 225 μg L^{-1} at 60°C. They also tested the effect of pH on the solubility at 12°C, but from the measured values it is difficult to indentify any impact of pH as large experimental errors are apparent. Liu and Lee[46] determined the solubility of 8:2 FTOH by direct aqueous phase measurements and a log-linear cosolvency approach. The cosolvent-extrapolated water solubility is 0.224 mg L^{-1}, in good agreement with the directly measured value of 0.194 mg L^{-1} from water. Using the two methods, Liu and Lee[47] measured the solubilities of 4:2 FTOH, 6:2 FTOH and 10:2 FTOH. The average measured water solubility for 4:2 FTOH and 6:2 FTOH is 974 mg L^{-1} and 18.8 mg L^{-1}, respectively. Estimation of the water solubility of 10:2 FTOH yielded a value of 0.011 mg L^{-1}. Single log-linear correlations between aqueous solubility and modified McGowan molar volumes were obtained for the n-alkanols and FTOHs. Each CF_2 moiety decreased the aqueous solubility by ~0.78 log units (compared to 0.60 log units for each CH_2 addition in hydrogenated primary alcohols). The perfluorocarbon chain length was found to be the dominant structural feature influencing solubility. Fischer-Drowos et al.[48] determined the solubility of 2H-hexadecafluoro-2-decenoic acid (8:2 FTUCA) using a LC-MS/MS

TABLE 2. Predicted water solubility of some environmentally relevant PFCs at 25°C

Chemical	CAS	Sw (mg L^{-1})	Method	Reference
PFBA	375-22-4	4.47×10^{2}	QSPR	49
PFPeA	2706-90-3	1.20×10^{2}	QSPR	49
PFHxA	307-24-4	2.95×10	QSPR	49
PFHpA	375-85-9	6.61	QSPR	49
PFOA	335-67-1	1.74	QSPR	49
PFNA	375-95-1	1.8×10^{-1}	QSPR	49
PFDA	335-76-2	2.8×10^{-2}	QSPR	49
PFUnA	2058-94-8	1.5×10^{-3}	QSPR	49
PFDoA	307-55-1	7.59×10^{-5}	QSPR	49
PFTriA	72629-94-8	2.51×10^{-6}	QSPR	49
4:2 FTOH	2043-47-2	8.71×10	QSPR	49
6:2 FTOH	647-42-7	7.41	QSPR	49
8:2 FTOH	678-39-7	2.4×10^{-1}	QSPR	49
10:2 FTOH	865-86-1	6.9×10^{-4}	QSPR	49
PFHxS	355-46-4	7.59	QSPR	49
PFOS	1763-23-1	2.1×10^{-1}	QSPR	49
PFOSA	754-91-6	2.4×10^{-1}	QSPR	49
N-MeFOSE	24448-09-7	7.2×10^{-1}	QSPR	49
N-EtFOSE	1691-99-2	8.7×10^{-1}	QSPR	49
N-EtFOSEA	423-82-5	1.38	QSPR	49

system. An aqueous solubility of 64 ± 5 mg L^{-1} at ambient temperatures was observed.

Experimental measurements of water solubility are cost and time-consuming, and require solving questions with regard to synthesis and analysis of samples. As an alternative to experimental assessment, Bhhatarai and Gramatica[49] developed quantitative structure-property relationships (QSPR) for the water solubility of PFCs based on experimental data of 20 PFCs. The authors predicted water solubility of a set of 154 PFCs using the QSPR models developed. The predicted water solubility for some environmentally relevant PFCs is shown in Table 2.

Vapor Pressure

Vapor pressure is an important parameter for determining the partition behavior of a chemical between the air phase and the liquid/solid phase. Vapor pressure is of use in assessing the ability of a chemical to partition into the gas phase and the potential of the compound for long-range transport to remote locations via air. Accurate assessment of vapor pressure is especially important for precursors of perfluoroalkyl acids (PFAAs), as they have been reported to be the source of PFOA/PFOS in remote locations, such as the Arctic area.

Crowder et al.[50] determined vapor pressures (up to approximately 1 atm) of 10 PFCs: C_3F_8, n-C_5F_{12}, 2-CF_3-C_4F_9, n-C_6F_{14} (perfluoro-n-hexane),

2-CF_3-C_5F_{11}, 3-CF_3-C_5F_{11}, 2,3-$(CF_3)_2$-C_4F_8, cyclo-C_5F_{10}, and cyclo-C_6F_{12}, 1,2-$(CF_3)_2$-cyclo-C_4F_6, which were purified by gas chromatography before measurement. Mousat[51] measured the vapor pressure of perfluoro-*n*-hexane from 100°C to the critical point, and found that vapor pressure could be predicted well by a two-parameter equation. When the same vapor pressure equation was applied to the data reported by Crowder et al.[50] agreement between experimental and calculated values with less than 6% deviation was obtained. Dias et al.[52] measured vapor pressures of perfluoro-*n*-hexane, perfluoro-n-nonane, perfluorodecalin, 1H-perfluoro-n-octane, perfluorobenzene, and perfluorotoluene in the temperature range of 288 to 333 K with an apparatus based on the static method. Steele et al.[53,54] measured vapor pressures of pentafluorophenol and perfluoro-n-heptane to a pressure limit of 270 kPa using a twin ebulliometric apparatus (Bartlesville, OK, USA), and fitted the measured data with the Wagner vapor pressure equation.

For PFAAs, Steele et al.[55,56] determined the vapor pressures of perfluoroheptanoic acid (PFHpA) and perfluorobutanoic acid (PFBA) to a pressure limit of 270 kPa using the same twin ebulliometric apparatus, and also fitted the data with the Wagner vapor pressure equation. Kaiser et al.[57] determined the vapor pressures of perfluorooctanoic, -nonanoic, -decanoic, -undecanoic, and -dodecanoic acids using a dynamic method developed by Scott.[58] Subsequently, the temperature-dependent vapor pressure data were fitted with the Antoine equation. Washburn et al.[59] measured the vapor pressures of PFOA and APFO using the same dynamic measurement procedure developed by Scott[58] and a gas saturation method from the U.S. Environmental Protection Agency, respectively, and obtained Antoine equations for these two chemicals. Barton et al.[60] determined the vapor pressure of PFOA at ambient temperatures using a modified gas saturation method. The vapor pressures obtained compared favorably with subcooled vapor pressures extrapolated from the Antoine equation presented in the paper of Kaiser et al.[57] Using the same method, Barton et al.[61] measured the solid vapor pressure of ammonium perfluorooctanoate (APFO) over the temperature range of 45~60°C. After fitting the data into the Clausius-Clayperon equation, these authors calculated the ambient vapor pressure of APFO at 25°C to be 0.003 Pa, which was three orders of magnitude lower than that of PFOA.

As FTOHs are recognized as volatile precursors of PFCAs and have the potential to be long-range transported via atmosphere, they are of special concern. Kaiser et al.[45] measured the vapor pressure of 8:2 FTOH in the temperature range of 21–201°C using three different methods, including a novel method based on gas-phase NMR. The result from the single measurement taken at 21°C with the gas saturation method was 3 Pa for 8:2 FTOH. The extrapolated value based on a linear least-square fit of the Scott data was 15 Pa, which is slightly higher than the value (7 Pa) obtained by the Antoine equation at the same temperature.

Lei et al.[62] estimated the liquid-phase vapor pressures of 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, 10:2 FTOH, N-ethyl perfluorooctane sulfonamide (N-EtFOSA), N-methyl perfluorooctane sulfonamidoethanol (N-MeFOSE), and N-ethyl perfluorooctane sulfonamidoethanol (N-EtFOSE), as a function of temperature using a technique based on gas chromatographic retention times relative to those of hexachlorobenzene. The method was calibrated using volatility data for fluorinated aromatic substances, chlorinated benzenes, and pesticides. The fluorinated telomer alcohols were found to have a volatility that was higher than that of the nonfluorinated alcohols of similar chain length, and higher than that of perfluorinated aromatics of comparable molar mass. On the basis of their volatility, the polyfluorinated chemicals are expected to occur predominantly in the atmospheric gas phase. Shoeib et al.[63] determined the solid-phase vapor pressures (Ps) of N-MeFOSE, N-EtFOSE, and N-MeFOSEA at room temperature (23°C) using the generator column method described by Wania et al.[64] The Ps values were measured as 4.0×10^{-4} Pa, 1.7×10^{-3} Pa, and 4.1×10^{-4} Pa, respectively.

Stock et al.[65] measured the vapor pressures of 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH using the boiling point method, and found that the measured values were similar to those of Lei et al.[62] When comparing their findings with literature data, Stock et al.[65] found that perfluorocarbons (C2-C8) and the fluorinated telomer alcohols researched have vapor pressures equal to or greater than that of their hydrogen analogues. After analyses, the authors suggested that this is due to the unique geometry of PFCs—in particular the stiff, helical perfluorinated chain and the significant intramolecular hydrogen bonding of the FTOHs.

Krusic et al.[66] determined the vapor pressures of 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH using three independent methods: (a) a method suitable for very low vapor pressures at ambient temperatures (gas-saturation method), (b) an improved boiling point method at controlled pressures (Scott method), and (c) a novel method, requiring milligram quantities of substance, based on gas-phase NMR (Wilmington, DE, USA). Their results at ambient temperature are significantly lower than those reported by Lei et al.[62] and Stock et al.[65] After the use of gas-phase NMR, gas-phase FTIR, 2D NMR heteronuclear Overhauser effect measurements, high-level *ab initio* computations were used to investigate the intramolecular hydrogen bonding in fluorotelomer alcohols. It was pointed out that intramolecular hydrogen bonding in these chemicals is very weak and cannot contribute to or cause unusual volatility.

Goss et al.[67] compared the liquid vapor pressures of *n*-alkanes, *n*-alcohols, *n*-perfluoroalkane, and FTOHs in a natural logarithm plot of vapor pressures versus the number of C-atoms. These authors postulated that homologue compound classes in the plot must result in parallel lines as they grow by the same molecular fragment ($-CH_2-$ or $-CF_2-$), which adds

an identical increment to the overall interaction energy. In addition, 10:2 FTOH should not have a vapor pressure that is even higher than that of the *n*-perfluoroalkane or hydrocarbon analogue. They therefore declared that the data of Lei et al.[62] and Stock et al.[65] are not plausible, while the data from Krusic et al.[66] agree to these expectations. Furthermore it was pointed out that the main problem regarding the findings of Lei et al.[62] is that retention on a nonpolar DB-1 column that cannot form H-bonds with any analyte is not a suitable measure for the vapor pressure of compounds that can form strong intermolecular H-bonds in their pure liquid phase. With respect to the measurement errors of Stock et al.,[65] it was speculated that these errors arise from the linear extrapolation of their experimental data over a temperature difference of more than 100°C. The heat of vaporization cannot be expected to stay constant over such a large temperature interval.

Based on the vapor pressures reported by Shoeib et al.,[63] Krusic et al.,[66] and Kaiser et al.,[57] Arp et al.[68] evaluated the performances of four models, and found that COSMOtherm (COSMOlogic GmbH & Co. KG, Leverkusen, Germany) predictions agree within one order of magnitude with experimental values, while SPARC (University of Georgia, Athens, GA, USA) and EPI Suite (US EPA) perform considerably worse. In the Supporting Information, Arp et al. provided predicted vapor pressures for some PFCs, including PFCAs, FTOHs, and fluorotelomer olefins. Recognizing these problems, SPARC and EPI Suite have been updated, but the software has not likely been more broadly altered to better capture the estimation ability of all perfluorinated compounds.[3]

Cobranchi et al.[69] used two distinctly different capillary gas chromatographic methods to determine the vapor pressure of 8:2 FTOH and 1-H perfluoroheptane at several temperatures. It was found that vapor pressure estimated by the relative retention method could differ by as much as one order of magnitude compared to published results determined by other (nonchromatographic) methods. The authors pointed out that this variance may be a function of solvent-solute interactions within the gas chromatographic column and the infinite dilution assumption. In addition, it was discussed that the results of headspace gas chromatography (GC) with an atomic emission detection (AED) method were consistent with prior nonchromatographic results, as the former method can avoid the problems depicted previously.

Table 3 provides an overview of experimentally determined vapor pressures of PFCs reported in literature at various temperatures, whereas Table 4contains reported vapor pressures of PFCs at 25°C.

Bhhatarai and Gramatica[49] also developed a QSPR model for vapor pressures of PFCs based on experimental data of 24 PFCs. The authors predicted vapor pressures of another set of 150 PFCs using the QSPR model developed. The calculated vapor pressures for some environmentally relevant PFCs are included in Table 4.

TABLE 3. Overview of vapor pressures of PFCs

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
Perfluoropropane (L)	181.77	3.52	the pressure gauge	50
	195.12	3.98	the pressure gauge	50
	211.79	4.45	the pressure gauge	50
	217.74	4.59	the pressure gauge	50
	224.28	4.75	the pressure gauge	50
	229.76	4.86	the pressure gauge	50
	235.26	4.98	the pressure gauge	50
	237.34	5.03	the pressure gauge	50
Perfluoro-n-pentane (L)	221.17	3.09	the pressure gauge	50
	226.36	3.27	the pressure gauge	50
	240.16	3.69	the pressure gauge	50
	250.66	3.96	the pressure gauge	50
	259.91	4.18	the pressure gauge	50
	271.76	4.44	the pressure gauge	50
	280.7	4.63	the pressure gauge	50
	286.13	4.73	the pressure gauge	50
	289.75	4.79	the pressure gauge	50
	294.25	4.87	the pressure gauge	50
	297.19	4.92	the pressure gauge	50
	297.59	4.93	the pressure gauge	50
	302.32	5.01	the pressure gauge	50
	303	5.02	the pressure gauge	50
Perfluoro-n-hexane (L)	256.43	3.52	the pressure gauge	50
	261.63	3.64	the pressure gauge	50
	267.69	3.80	the pressure gauge	50
	282.77	4.16	the pressure gauge	50
	291.52	4.34	the pressure gauge	50
	307.07	4.63	the pressure gauge	50
	311.24	4.71	the pressure gauge	50
	315.68	4.78	the pressure gauge	50
	319.37	4.84	the pressure gauge	50
	324.85	4.92	the pressure gauge	50
	330.35	5.01	the pressure gauge	50
	333.59	5.05	the pressure gauge	50
	332.99	5.05	the pressure gauge	50
	341.1	5.17	the pressure gauge	50
	351.85	5.31	the pressure gauge	50
	374.78	5.57	the pressure gauge	50
	395.56	5.79	the pressure gauge	50
	415.48	5.97	the pressure gauge	50
	432.68	6.11	the pressure gauge	50
	447.08	6.23	the pressure gauge	50
	433.42	6.13	the pressure gauge	51
	434.67	6.14	the pressure gauge	51
	435.54	6.15	the pressure gauge	51
	436.58	6.16	the pressure gauge	51
	438.5	6.18	the pressure gauge	51
	439.99	6.19	the pressure gauge	51
	441.27	6.21	the pressure gauge	51
	442.57	6.22	the pressure gauge	51
	444.01	6.23	the pressure gauge	51

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	445.88	6.25	the pressure gauge	51
	447.46	6.26	the pressure gauge	51
	448.77	6.27	the pressure gauge	51
	288.31	4.27	the static method	52
	293.22	4.37	the static method	52
	298.09	4.47	the static method	52
	302.98	4.56	the static method	52
	307.97	4.65	the static method	52
	313.17	4.74	the static method	52
	318.15	4.82	the static method	52
	323.13	4.90	the static method	52
	328.11	4.97	the static method	52
	333.1	5.05	the static method	52
Perfluoro-n-heptane	303.677	4.12	a twin ebulliometric apparatus n-decane (r)	54
	308.468	4.22	a twin ebulliometric apparatus n-decane (r)	54
	312.428	4.30	a twin ebulliometric apparatus n-decane (r)	54
	317.683	4.40	a twin ebulliometric apparatus n-decane (r)	54
	317.675	4.40	a twin ebulliometric apparatus water (r)	54
	322.988	4.49	a twin ebulliometric apparatus water (r)	54
	328.292	4.59	a twin ebulliometric apparatus water (r)	54
	333.676	4.68	a twin ebulliometric apparatus water (r)	54
	339.073	4.76	a twin ebulliometric apparatus water (r)	54
	344.553	4.85	a twin ebulliometric apparatus water (r)	54
	350.037	4.93	a twin ebulliometric apparatus water (r)	54
	355.586	5.01	a twin ebulliometric apparatus water (r)	54
	361.174	5.08	a twin ebulliometric apparatus water (r)	54
	366.806	5.16	a twin ebulliometric apparatus water (r)	54
	372.475	5.23	a twin ebulliometric apparatus water (r)	54
	378.184	5.30	a twin ebulliometric apparatus water (r)	54
	383.928	5.37	a twin ebulliometric apparatus water (r)	54
	389.712	5.43	a twin ebulliometric apparatus water (r)	54
Perfluoro-n-nonane	288.18	2.83	the static method	52
	293.18	2.97	the static method	52
	298.11	3.11	the static method	52

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	303.1	3.23	the static method	52
	308.19	3.37	the static method	52
	313.17	3.49	the static method	52
	318.22	3.60	the static method	52
	323.15	3.71	the static method	52
	328.15	3.82	the static method	52
	333.15	3.93	the static method	52
Perfluoro-2-methylbutane (L)	228.7	3.32	the pressure gauge	50
	235.93	3.55	the pressure gauge	50
	253.05	4.01	the pressure gauge	50
	261.25	4.20	the pressure gauge	50
	273.59	4.47	the pressure gauge	50
	282.81	4.65	the pressure gauge	50
	301.84	4.98	the pressure gauge	50
	305.28	5.04	the pressure gauge	50
Perfluoro-2-methylpentane (L)	253.93	3.43	the pressure gauge	50
	266.54	3.77	the pressure gauge	50
	276.49	4.01	the pressure gauge	50
	280.72	4.11	the pressure gauge	50
	288.39	4.27	the pressure gauge	50
	293.74	4.38	the pressure gauge	50
	298.86	4.48	the pressure gauge	50
	304.28	4.58	the pressure gauge	50
	307.88	4.64	the pressure gauge	50
	311.08	4.70	the pressure gauge	50
	312.09	4.71	the pressure gauge	50
	314.54	4.76	the pressure gauge	50
	319.37	4.83	the pressure gauge	50
	325.43	4.93	the pressure gauge	50
	328.44	4.97	the pressure gauge	50
	332.08	5.02	the pressure gauge	50
	333.23	5.05	the pressure gauge	50
	351.65	5.30	the pressure gauge	50
	370.65	5.52	the pressure gauge	50
	417.39	5.97	the pressure gauge	50
	433.13	6.10	the pressure gauge	50
	441.7	6.17	the pressure gauge	50
	447.54	6.22	the pressure gauge	50
	449.41	6.23	the pressure gauge	50
	451.11	6.25	the pressure gauge	50
Perfluoro-3-methylpentane (L)	255.15	3.48	the pressure gauge	50
	262.33	3.67	the pressure gauge	50
	264.01	3.72	the pressure gauge	50
	268.86	3.84	the pressure gauge	50
	277.48	4.03	the pressure gauge	50
	282.79	4.15	the pressure gauge	50

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	288.55	4.27	the pressure gauge	50
	294.27	4.38	the pressure gauge	50
	300.78	4.51	the pressure gauge	50
	308.67	4.65	the pressure gauge	50
	313.66	4.73	the pressure gauge	50
	319.28	4.82	the pressure gauge	50
	323.87	4.89	the pressure gauge	50
	329.38	4.98	the pressure gauge	50
	332.22	5.02	the pressure gauge	50
	342.04	5.17	the pressure gauge	50
	351.45	5.29	the pressure gauge	50
	376.72	5.58	the pressure gauge	50
	394.73	5.76	the pressure gauge	50
	417.05	5.96	the pressure gauge	50
	432.99	6.09	the pressure gauge	50
	448.56	6.22	the pressure gauge	50
	449.78	6.22	the pressure gauge	50
Perfluoro-2,3-dimethylbutan (L)	262.33	3.64	the pressure gauge	50
	270.34	3.84	the pressure gauge	50
	274.67	3.94	the pressure gauge	50
	278.16	4.01	the pressure gauge	50
	283.72	4.14	the pressure gauge	50
	288.9	4.25	the pressure gauge	50
	293.41	4.33	the pressure gauge	50
	296.91	4.41	the pressure gauge	50
	300.26	4.47	the pressure gauge	50
	303.97	4.53	the pressure gauge	50
	308.78	4.62	the pressure gauge	50
	313.75	4.70	the pressure gauge	50
	318.61	4.78	the pressure gauge	50
	323.3	4.86	the pressure gauge	50
	328.38	4.94	the pressure gauge	50
	332.63	5.00	the pressure gauge	50
	341.28	5.12	the pressure gauge	50
	351.21	5.25	the pressure gauge	50
	354.63	5.52	the pressure gauge	50
	394.65	5.73	the pressure gauge	50
	414.62	5.90	the pressure gauge	50
	433.22	6.06	the pressure gauge	50
	450.36	6.20	the pressure gauge	50
	451.76	6.21	the pressure gauge	50
	452.79	6.22	the pressure gauge	50
Perfluorocyclopentane (L)	285.6	4.84	the pressure gauge	50
	290.56	4.92	the pressure gauge	50
	296.83	5.02	the pressure gauge	50
Perfluorocyclopentane (S)	229.67	3.42	the pressure gauge	50
	251.21	4.02	the pressure gauge	50
	262.42	4.31	the pressure gauge	50

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	272.8	4.56	the pressure gauge	50
	281.17	4.75	the pressure gauge	50
Perfluorocyclohexane (L)	350.32	5.37	the pressure gauge	50
	378.36	5.68	the pressure gauge	50
	410.81	5.98	the pressure gauge	50
	433.17	6.16	the pressure gauge	50
	451.01	6.30	the pressure gauge	50
Perfluorocyclohexane (S)	252.7	3.33	the pressure gauge	50
	270.79	3.82	the pressure gauge	50
	283.92	4.15	the pressure gauge	50
	300.74	4.53	the pressure gauge	50
	307.85	4.67	the pressure gauge	50
	315.32	4.82	the pressure gauge	50
	319.05	4.88	the pressure gauge	50
	323.09	4.96	the pressure gauge	50
	325.82	5.00	the pressure gauge	50
Perfluoro-1,2-dimethylcyclobutan (L)	242.61	3.42	the pressure gauge	50
	248.15	3.59	the pressure gauge	50
	257.14	3.83	the pressure gauge	50
	264.64	4.01	the pressure gauge	50
	267.86	4.09	the pressure gauge	50
	273.36	4.21	the pressure gauge	50
	276.98	4.29	the pressure gauge	50
	283.31	4.41	the pressure gauge	50
	289.51	4.53	the pressure gauge	50
	299.85	4.72	the pressure gauge	50
	304.26	4.79	the pressure gauge	50
	309.06	4.87	the pressure gauge	50
	313.93	4.95	the pressure gauge	50
	318.01	5.01	the pressure gauge	50
1-H perfluoroheptane	318.15	3.59	Headspace GC/AED	69
	328.15	3.76	Headspace GC/AED	69
	338.15	3.91	Headspace GC/AED	69
	348.15	4.06	Headspace GC/AED	69
	358.15	4.19	Headspace GC/AED	69
1H-Perfluoro-n-octane	288.35	3.07	the static method	52
	293.26	3.20	the static method	52
	298.26	3.34	the static method	52
	303.19	3.45	the static method	52
	308.15	3.57	the static method	52
	313.13	3.69	the static method	52
	318.06	3.79	the static method	52
	323.01	3.91	the static method	52
	328	4.01	the static method	52
	332.89	4.11	the static method	52
Perfluorobenzene	288.23	3.84	the static method	52
	293.51	3.95	the static method	52
	297.98	4.05	the static method	52

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	302.96	4.15	the static method	52
	307.96	4.25	the static method	52
	312.96	4.35	the static method	52
	317.96	4.44	the static method	52
	322.96	4.53	the static method	52
	327.95	4.61	the static method	52
	333.01	4.70	the static method	52
Perfluorotoluene	288.23	3.34	the static method	52
	293.2	3.47	the static method	52
	298.28	3.59	the static method	52
	303.18	3.71	the static method	52
	308.15	3.82	the static method	52
	313.15	3.93	the static method	52
	318.15	4.03	the static method	52
	323.15	4.13	the static method	52
	328.45	4.23	the static method	52
	333.93	4.33	the static method	52
Pentafluorophenol	323.051	3.30	a twin ebulliometric apparatus n-decane (r)	53
	335.747	3.60	a twin ebulliometric apparatus n-decane (r)	53
	341.373	3.73	a twin ebulliometric apparatus n-decane (r)	53
	349.788	3.90	a twin ebulliometric apparatus n-decane (r)	53
	356.071	4.03	a twin ebulliometric apparatus n-decane (r)	53
	361.183	4.12	a twin ebulliometric apparatus n-decane (r)	53
	366.488	4.22	a twin ebulliometric apparatus n-decane (r)	53
	370.855	4.30	a twin ebulliometric apparatus n-decane (r)	53
	376.682	4.40	a twin ebulliometric apparatus n-decane (r)	53
	376.678	4.40	a twin ebulliometric apparatus water (r)	53
	382.508	4.49	a twin ebulliometric apparatus water (r)	53
	388.372	4.59	a twin ebulliometric apparatus water (r)	53
	394.265	4.68	a twin ebulliometric apparatus water (r)	53
	400.214	4.76	a twin ebulliometric apparatus water (r)	53
	406.191	4.85	a twin ebulliometric apparatus water (r)	53
	412.206	4.93	a twin ebulliometric apparatus water (r)	53
	418.258	5.01	a twin ebulliometric apparatus water (r)	53

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	424.354	5.08	a twin ebulliometric apparatus water (r)	53
	430.481	5.16	a twin ebulliometric apparatus water (r)	53
	436.655	5.23	a twin ebulliometric apparatus water (r)	53
	442.875	5.30	a twin ebulliometric apparatus water (r)	53
	449.119	5.37	a twin ebulliometric apparatus water (r)	53
	455.414	5.43	a twin ebulliometric apparatus water (r)	53
PFBA	310.883	3.30	a twin ebulliometric apparatus n-decane (r)	56
	322.259	3.60	a twin ebulliometric apparatus n-decane (r)	56
	327.331	3.73	a twin ebulliometric apparatus n-decane (r)	56
	334.848	3.90	a twin ebulliometric apparatus n-decane (r)	56
	340.416	4.03	a twin ebulliometric apparatus n-decane (r)	56
	344.952	4.12	a twin ebulliometric apparatus n-decane (r)	56
	349.616	4.22	a twin ebulliometric apparatus n-decane (r)	56
	353.48	4.30	a twin ebulliometric apparatus n-decane (r)	56
	358.572	4.40	a twin ebulliometric apparatus n-decane (r)	56
	363.666	4.49	a twin ebulliometric apparatus water (r)	56
	368.782	4.59	a twin ebulliometric apparatus water (r)	56
	373.916	4.68	a twin ebulliometric apparatus water (r)	56
	379.058	4.76	a twin ebulliometric apparatus water (r)	56
	384.231	4.85	a twin ebulliometric apparatus water (r)	56
	389.41	4.93	a twin ebulliometric apparatus water (r)	56
	394.628	5.01	a twin ebulliometric apparatus water (r)	56
	399.854	5.08	a twin ebulliometric apparatus water (r)	56
	405.091	5.16	a twin ebulliometric apparatus water (r)	56
	410.352	5.23	a twin ebulliometric apparatus water (r)	56
	415.632	5.30	a twin ebulliometric apparatus water (r)	56

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	420.94	5.37	a twin ebulliometric apparatus water (r)	56
	426.264	5.43	a twin ebulliometric apparatus water (r)	56
PFHpA	359.074	3.30	a twin ebulliometric apparatus n-decane (r)	55
	371.394	3.60	a twin ebulliometric apparatus n-decane (r)	55
	376.901	3.73	a twin ebulliometric apparatus n-decane (r)	55
	385.002	3.90	a twin ebulliometric apparatus n-decane (r)	55
	391.081	4.03	a twin ebulliometric apparatus n-decane (r)	55
	395.927	4.12	a twin ebulliometric apparatus n-decane (r)	55
	400.977	4.22	a twin ebulliometric apparatus n-decane (r)	55
	405.187	4.30	a twin ebulliometric apparatus n-decane (r)	55
	410.691	4.40	a twin ebulliometric apparatus n-decane (r)	55
	410.705	4.40	a twin ebulliometric apparatus water (r)	55
	416.226	4.49	a twin ebulliometric apparatus water (r)	55
	421.789	4.59	a twin ebulliometric apparatus water (r)	55
	427.369	4.68	a twin ebulliometric apparatus water (r)	55
	432.985	4.76	a twin ebulliometric apparatus water (r)	55
	438.648	4.85	a twin ebulliometric apparatus water (r)	55
	444.318	4.93	a twin ebulliometric apparatus water (r)	55
	450.03	5.01	a twin ebulliometric apparatus water (r)	55
	455.777	5.08	a twin ebulliometric apparatus water (r)	55
	461.56	5.16	a twin ebulliometric apparatus water (r)	55
	467.373	5.23	a twin ebulliometric apparatus water (r)	55
	473.223	5.30	a twin ebulliometric apparatus water (r)	55
	479.111	5.37	a twin ebulliometric apparatus water (r)	55
	485.03	5.43	a twin ebulliometric apparatus water (r)	55
PFOA	332.4	2.11	a dynamic method	57
	335.52	2.23	a dynamic method	57

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	339.75	2.36	a dynamic method	57
	345.75	2.56	a dynamic method	57
	352.15	2.76	a dynamic method	57
	359.75	2.98	a dynamic method	57
	368.05	3.20	a dynamic method	57
	377.09	3.43	a dynamic method	57
	387.15	3.66	a dynamic method	57
	397.55	3.89	a dynamic method	57
	409.26	4.12	a dynamic method	57
	421.75	4.35	a dynamic method	57
	435.35	4.57	a dynamic method	57
	450.95	4.81	a dynamic method	57
	463.95	4.98	a dynamic method	57
	332.4	2.11	Scott method	59
	335.52	2.23	Scott method	59
	339.75	2.36	Scott method	59
	345.75	2.56	Scott method	59
	352.15	2.76	Scott method	59
	359.75	2.98	Scott method	59
	368.05	3.20	Scott method	59
	377.09	3.43	Scott method	59
	387.15	3.66	Scott method	59
	397.55	3.89	Scott method	59
	409.26	4.12	Scott method	59
	421.75	4.35	Scott method	59
	435.35	4.57	Scott method	59
	450.95	4.81	Scott method	59
	463.95	4.98	Scott method	59
	300.25 (*S*)	0.72	a modified gas saturation method	60
	308.55 (*S*)	1.26	a modified gas saturation method	60
	318.85 (*S*)	1.61	a modified gas saturation method	60
APFO	363.24	0.57	a gas saturation method	59
	372.82	0.98	a gas saturation method	59
	382.57	1.32	a gas saturation method	59
	392.33	1.64	a gas saturation method	59
	423.15	2.90	a gas saturation method	59
	433.15	3.30	a gas saturation method	59
	438.15	3.47	a gas saturation method	59
	318.55	−1.40	a modified gas saturation method	61
	328.65	−1.05	a modified gas saturation method	61
	333.55	−0.77	a modified gas saturation method	61
PFNA (L)	372.78	3.05	a dynamic method	57
	382.69	3.31	a dynamic method	57
	390.81	3.49	a dynamic method	57
	401.7	3.74	a dynamic method	57
	412.92	3.97	a dynamic method	57

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	423.86	4.18	a dynamic method	57
	432.85	4.36	a dynamic method	57
	446.11	4.57	a dynamic method	57
	461.08	4.79	a dynamic method	57
	476.13	5.00	a dynamic method	57
	476.27	5.00	a dynamic method	57
PFDA (L)	402.71	3.50	a dynamic method	57
	406.24	3.57	a dynamic method	57
	414.08	3.74	a dynamic method	57
	425.35	3.97	a dynamic method	57
	436.83	4.18	a dynamic method	57
	447.12	4.36	a dynamic method	57
	460.65	4.57	a dynamic method	57
	476.08	4.79	a dynamic method	57
	492.03	5.00	a dynamic method	57
PFUnA (L)	385.19	2.79	a dynamic method	57
	393.78	3.03	a dynamic method	57
	402.69	3.24	a dynamic method	57
	412.29	3.44	a dynamic method	57
	412.41	3.45	a dynamic method	57
	422.37	3.65	a dynamic method	57
	431.47	3.84	a dynamic method	57
	445.17	4.09	a dynamic method	57
	459.92	4.32	a dynamic method	57
	473.83	4.54	a dynamic method	57
	475.24	4.54	a dynamic method	57
	491.44	4.77	a dynamic method	57
	491.79	4.77	a dynamic method	57
	509.83	4.98	a dynamic method	57
	510.8	5.00	a dynamic method	57
PFDoA (L)	400.73	2.93	a dynamic method	57
	413.72	3.26	a dynamic method	57
	422.1	3.46	a dynamic method	57
	422.75	3.47	a dynamic method	57
	433.27	3.69	a dynamic method	57
	445.63	3.92	a dynamic method	57
	457.94	4.14	a dynamic method	57
	470.59	4.34	a dynamic method	57
	483.66	4.54	a dynamic method	57
	498.85	4.74	a dynamic method	57
	520.51	5.00	a dynamic method	57
4:2 FTOH	298.15	3.00	the boiling point method	65
6:2 FTOH	298.15	2.87	the boiling point method	65
8:2 FTOH	298.15	2.39	the boiling point method	65
10:2 FTOH	298.15	2.13	the boiling point method	65
4:2 FTOH	298.15	3.22	GC-retention time	62
6:2 FTOH	298.15	2.94	GC-retention time	62
8:2 FTOH	298.15	2.36	GC-retention time	62
10:2 FTOH	298.15	1.72	GC-retention time	62
N-EtFOSA	298.15	0.85	GC-retention time	62
N-MeFOSE	298.15	−0.15	GC-retention time	62
N-EtFOSE	298.15	−0.46	GC-retention time	62

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
N-MeFOSE	296.15	−3.40	the generator column method	63
N-EtFOSE	296.15	−2.77	the generator column method	63
N-MeFOSEA	296.15	−3.39	the generator column method	63
4:2 FTOH	323.15	3.08	gas-phase NMR	66
	347.95	3.81	gas-phase NMR	66
	372.35	4.34	gas-phase NMR	66
	382.05	4.52	gas-phase NMR	66
	391.75	4.71	gas-phase NMR	66
	401.45	4.86	gas-phase NMR	66
	406.35	4.94	gas-phase NMR	66
	411.15	5.00	gas-phase NMR	66
	415.95	5.08	gas-phase NMR	66
	420.75	5.15	gas-phase NMR	66
6:2 FTOH	308.15	2.03	gas-saturation method	66
	333.95	2.81	Scott method	66
	345.65	3.15	Scott method	66
	347.95	3.08	gas-phase NMR	66
	358.35	3.48	Scott method	66
	370.95	3.76	Scott method	66
	372.35	3.70	gas-phase NMR	66
	383.05	4.02	Scott method	66
	394.45	4.24	Scott method	66
	396.65	4.20	gas-phase NMR	66
	409.15	4.49	Scott method	66
	419.35	4.65	Scott method	66
	420.75	4.59	gas-phase NMR	66
	430.35	4.78	gas-phase NMR	66
	431.25	4.83	Scott method	66
	439.95	4.91	gas-phase NMR	66
	444.45	5.00	Scott method	66
	449.45	5.04	gas-phase NMR	66
8:2 FTOH	294.15 (*S*)	0.48	gas-saturation method	66
	357.15	2.85	Scott method	66
	369.15	3.18	Scott method	66
	372.35	3.23	gas-phase NMR	66
	380.15	3.45	Scott method	66
	392.15	3.69	Scott method	66
	396.65	3.76	gas-phase NMR	66
	403.15	3.92	Scott method	66
	415.15	4.15	Scott method	66
	420.75	4.23	gas-phase NMR	66
	428.15	4.36	Scott method	66
	439.15	4.53	Scott method	66
	444.75	4.60	gas-phase NMR	66
	452.15	4.72	Scott method	66
	459.05	4.79	gas-phase NMR	66
	463.15	4.86	Scott method	66
	468.45	4.91	gas-phase NMR	66
	474.15	5.00	Scott method	66

(Continued on next page)

TABLE 3. Overview of vapor pressures of PFCs *(Continued)*

Chemical	Temperature (K)	Log P (Pa)	Method	Reference
	477.95	5.04	gas-phase NMR	66
	482.65	5.10	gas-phase NMR	66
	318.15	1.46	Headspace GC/AED	69
	323.15	1.60	Headspace GC/AED	69
	328.15	1.67	Headspace GC/AED	69
	333.15	1.90	Headspace GC/AED	69
8:2 FTOH	294.15	0.48	gas-saturation	45
	357.15	2.85	Scott method	45
	369.15	3.17	Scott method	45
	380.15	3.44	Scott method	45
	392.15	3.69	Scott method	45
	397.15	3.71	NMR spectroscopy	45
	403.15	3.92	Scott method	45
	415.15	4.14	Scott method	45
	421.15	4.18	NMR spectroscopy	45
	428.15	4.36	Scott method	45
	430.15	4.37	NMR spectroscopy	45
	439.15	4.54	Scott method	45
	440.15	4.51	NMR spectroscopy	45
	445.15	4.55	NMR spectroscopy	45
	452.15	4.71	Scott method	45
	463.15	4.86	Scott method	45
	468.15	4.88	NMR spectroscopy	45
	474.15	5.00	Scott method	45
	498.15	5.21	NMR spectroscopy	45
10:2 FTOH	308.15 (*S*)	0.15	gas-saturation method	66
	396.65	3.20	gas-phase NMR	66
	420.75	3.78	gas-phase NMR	66
	444.75	4.20	gas-phase NMR	66
	458.95	4.45	gas-phase NMR	66
	468.45	4.52	gas-phase NMR	66
	477.95	4.72	gas-phase NMR	66
	487.45	4.78	gas-phase NMR	66
	496.85	4.96	gas-phase NMR	66
	506.25	5.07	gas-phase NMR	66
	510.95	5.11	gas-phase NMR	66

p*K*a

The acid dissociation constant (*K*a) is the equilibrium constant for a chemical reaction known as dissociation in the acid-base conversion. Because of many orders of magnitude spanned by *K*a values, a logarithmic form is more commonly used in practice. As the dissociated and nondissociated species of an organic acid differ largely in their physicochemical behavior, p*K*a will determine the behavior of the organic acid at certain pH values in water, and thus p*K*a will influence the environmental fate of a chemical.

PFSAs are believed to be strong acids, which will be effectively ionized under all possible environmental conditions. More attention was therefore

TABLE 4. Vapor pressures of PFCs at 25°C

Chemical	Temperature (K)	P (Pa)	Method	Reference
PFBA	310.88~426.26	8.51×10^{2}	Calculated	56
PFHpA	359.07~485.03	2.07×10	Calculated	55
PFOA	332.4~463.95	4.19	Calculated	57
PFOA	300.25~318.85	4.14	Calculated	60
PFOA	332.4~463.5	4.19	Calculated	59
APFO	363.24~483.15	1.2×10^{-2}	Calculated	59
APFO	318.55~333.55	3×10^{-3}	Calculated	61
PFNA	372.78~476.27	1.27	Calculated	57
PFDA	402.71~492.03	2.3×10^{-1}	Calculated	57
PFUnA	385.19~509.83	1.0×10^{-1}	Calculated	57
PFDoA	400.73~520.51	—	Calculated	57
4:2 FTOH	333.15~398.15	9.92×10^{2}	Calculated	65
6:2 FTOH	—	7.13×10^{2}	Calculated	65
8:2 FTOH	—	2.54×10^{2}	Calculated	65
10:2 FTOH	413.15~498.15	1.44×10^{2}	Calculated	65
4:2 FTOH	303.15~353.15	1.67×10^{3}	GC-retention time	62
6:2 FTOH	303.15~353.15	8.76×10^{2}	GC-retention time	62
8:2 FTOH	303.15~353.15	2.27×10^{2}	GC-retention time	62
10:2 FTOH	303.15~353.15	5.3×10	GC-retention time	62
N-EtFOSA	303.15~353.15	7	GC-retention time	62
N-MeFOSE	303.15~353.15	7×10^{-1}	GC-retention time	62
N-EtFOSE	303.15~353.15	3.5×10^{-1}	GC-retention time	62
8:2 FTOH	294.15~498.15	6.88	Calculated	45
4:2 FTOH	323.15~420.75	2.14×10^{2}	NMR	66
6:2 FTOH	308.15~449.45	1.76×10	NMR	66
8:2 FTOH	294.15~482.15	3.77	NMR	66
10:2 FTOH	308.15~510.95	1.8×10^{-1}	NMR	66
4:2 Olefin		2.25×10^{4}	Calculated	68
6:2 Olefin		1.85×10^{3}	Calculated	68
8:2 Olefin		1.84×10^{2}	Calculated	68
10:2 Olefin		1.86×10	Calculated	68
12:2 Olefin		1.82	Calculated	68
4:2 FTOH		2.22×10^{2}	Calculated	68
6:2 FTOH		3.66×10	Calculated	68
8:2 FTOH		3.41	Calculated	68
10:2 FTOH		6.35×10^{-1}	Calculated	68
EtFOSA		9.19×10^{-2}	Calculated	68
MeFOSE		2.19×10^{-3}	Calculated	68
EtFOSE		3.86×10^{-3}	Calculated	68
MeFOSEA		1.88×10^{-4}	Calculated	68
PFHxA		1.14×10^{2}	Calculated	68
PFHpA		4.56×10	Calculated	68
PFOA		1.33×10	Calculated	68
PFNA		4.94	Calculated	68
PFDA		1.57	Calculated	68
PFUnA		6.77×10^{-1}	Calculated	68
PFDoA		2.01×10^{-1}	Calculated	68
PFOS		3.46	Calculated	68
PFOSA		1.03×10^{-1}	Calculated	68
PFBA	298.15 (L)	8.99×10^{2}	QSPR	49
PFPeA	298.15 (L)	3.42×10^{2}	QSPR	49

(Continued on next page)

TABLE 4. Vapor pressures of PFCs at 25°C *(Continued)*

Chemical	Temperature (K)	P (Pa)	Method	Reference
PFHxA	298.15 (L)	1.21×10^{2}	QSPR	49
PFHpA	298.15 (L)	3.93×10	QSPR	49
PFOA	298.15 (L)	1.21×10	QSPR	49
PFNA	298.15 (L)	3.50	QSPR	49
PFDA	298.15 (L)	1.01	QSPR	49
PFUnA	298.15 (L)	2.6×10^{-1}	QSPR	49
PFDoA	298.15 (L)	8.6×10^{-2}	QSPR	49
PFTriA	298.15 (L)	1.6×10^{-2}	QSPR	49
4:2 FTOH	298.15 (L)	2.78×10^{2}	QSPR	49
6:2 FTOH	298.15 (L)	2.21×10	QSPR	49
8:2 FTOH	298.15 (L)	1.64	QSPR	49
10:2 FTOH	298.15 (L)	1.3×10^{-1}	QSPR	49
PFHxS	298.15 (L)	3.12	QSPR	49
PFOS	298.15 (L)	3.2×10^{-1}	QSPR	49
PFOSA	298.15 (L)	8.8×10^{-2}	QSPR	49
N-MeFOSE	298.15 (L)	1.7×10^{-2}	QSPR	49
N-EtFOSE	298.15 (L)	9.0×10^{-3}	QSPR	49
N-EtFOSEA	298.15 (L)	2.9×10^{-4}	QSPR	49

put on assessing the p*K*a of PFCAs, as these are considered weak acids. Despite the fundamental importance of p*K*a, there are few experimental data and many controversies exist regarding the p*K*a of PFCAs. Moroi et al.[70] determined acidity constants of PFCAs by acid–base titration, electric conductivity, and solubility change with pH. For shorter PFCAs (C1 to C5), *K*a values increase from C1 to C3 and then decrease from C3 to C5, with an abrupt decrease at C5. For the intermediate PFCAs (C6 to C8), the *K*a values were not determined by the methods mentioned, because of the extreme difficulty in separating the colloidal acid particles from the aqueous phase as stated by these authors. For longer PFCAs (C9 to C11), *K*a values decrease with increasing alkyl chain length. In this case, *K*a values were determined from the solubility change upon changing solution pH. It was noted, however, that the measured PFCA acidity constants were not for the monomeric acid species, but for some undefined oligomeric species.

A hypothesis of aerosol-mediated water-air transfer of PFOA[71,72] states:

> Aqueous aerosols will be enriched in the anion (PFO) at the air-water interface, relative to their source water, because of its surfactant nature. When PFO is protonated at the surface of the water droplet, the neutral acid (PFOA) will readily partition to the gas phase.

Thus, the process will diminish the predicted rainout potential, and increase the long-range transport (LRT) potential of PFOA in the atmosphere via the gas phase. Recently, this hypothesis is under heavy debate. As this process is mainly driven by the remarkable surface activity of PFO and by the

magnitudes of p*K*a and *K*aw of PFOA, the uncertainty of p*K*a values of PFOA has been one of the issues of focus.

Recently, the p*K*a of PFOA has been reported and has been the subject of much debate.[3,71,73–80] Brace[81] measured a p*K*a of 2.80 for 0.005 M (2.07 mg L^{-1}) PFOA in 50% aqueous ethanol using NaOH as the titrant. Igarashi and Yotsuyanagi[82] determined a p*K*a of 1.01 for 0.015 M (6.21 mg L^{-1}) PFOA by titrating a series of ethanol/water solutions containing 0.1 M NaCl and then extrapolating a plot of p*K*a versus ethanol content to 0% ethanol. López-Fontán et al.[83] derived a p*K*a of 1.31 when they investigated the aggregation of sodium perfluorooctanoate in water. Goss[73,74] estimated that the p*K*a of PFOA should be close to zero based on analogy and molecular modeling, and presented p*K*a values of some highly fluorinated carboxylic acids calculated by SPARC (University of Georgia, Athens, GA, USA) and COSMO-RS (COSMOlogic GmbH & Co. KG, Leverkusen, Germany). Burns et al.[71] reported a p*K*a of 3.8 ± 0.1 for monomeric PFOA using a standard water-methanol mixed solvent approach. They stated that the acidity of PFOA is considerably weaker than the acidity of its shorter-chain PFCA homologues because of the differences in molecular and electronic structure, coupled with solvation effects. Goss and Arp[75] discussed some questions in the literature of Burns et al.,[71] pointed out that the correct p*K*a of monomeric PFOA is still not decided, and suggested to account for the wide range of reported pKa values from 0 to 4. Rayne and Forest[79,80] investigated the monomeric p*K*a values of PFCAs (C2∼C10) using semiempirical, *ab initio*, and density functional theory (DFT) studies, and stated that the monomeric p*K*a values of all PFCAs (both short- and long-chain, linear and branched) are less than 1, and most likely near or less than zero.

The debates about p*K*a are focused on (a) the influence of aggregation on p*K*a, (b) the existence of intramolecular hydrogen bonds, (c) the influence of carbon chain length and helical formation, and (4) the existence of sorption of PFCs. It is well known that PFCAs will easily aggregate, but the way PFCAs form aggregates and subsequently influence pKa is not determined. Burns et al.[71] reported a p*K*a of 3.8 ± 0.1 for monomeric PFOA and ∼2.3 for aggregated PFOA. They also suggested that a p*K*a correction factor should be applied to account for the concentration-dependent shift in acid–base equilibrium caused by aggregation. Rayne and Forest[80] claimed that there is no intramolecular hydrogen bonding in PFCAs from theoretical studies, and it is not possible for monomeric p*K*a of PFOA to be 3.8 as reported by Burns et al.[71] Burns et al.[71] stated that the unique chain-length dependent conformation of PFCAs alters their molecular orbital energies, and hence the electron densities at the site of proton-transfer on PFCAs, which leads to the observed change of p*K*a values of PFCAs. The rigid helical twist conformation of long-chain PFCAs mitigates their acidity, causing an increase in p*K*a for the monomeric species, and the fundamental conformational change renders the p*K*a to be different from the short chain

TABLE 5. Overview of p*K*a values of selected PFCs at 25°C

Chemical	p*K*a	Method	Reference
TFA	0.567	titration and electric conductivity	70
PFPrA	0.475	titration and electric conductivity	70
PFBA	0.394	titration and electric conductivity	70
PFPeA	0.569	titration and electric conductivity	70
PFHxA	0.840	titration and electric conductivity	70
PFOA	2.80	titration	81
PFOA	1.01	titration	82
PFOA	1.31	titration	83
PFOA	3.8	a water-methanol mixed solvent approach	71
PFOA	~0	estimated	73, 74
PFOA	~0	estimated	79, 80
PFNA	2.575	solubility change with pH	70
PFDA	2.606	solubility change with pH	70
PFUnA	3.128	solubility change with pH	70

PFCAs. The same authors also stated that the p*K*a values of monomeric perfluorononanoic acid (PFNA), PFDA, and the longer-chain PFCAs (>C10) are expected to be similar or slightly lower than those of PFOA as they are structurally similar. However, Rayne and Forest[79,80] showed that chain helicity is not expected to alter the structural or electronic characteristics of the carboxylate group, and helical PFCAs are not expected to have monomeric p*K*a values higher than the p*K*a value of trifluoroacetic acid (TFA). The cause of the increasing p*K*a values for longer chain PFCAs is presently unresolved.[3] Goss[73,74] pointed out that sorption to interfaces such as the water surface or the walls of a glass vessel may lead to an overestimation of the p*K*a value in the titration experiment. On the other hand, Burns et al.,[71] reported that sorption to glass was not detected within the time frame of their experiments conducted.

Some questions mentioned above are still unsolved, suggesting that future research efforts should be concentrated on the p*K*a of PFCAs, especially the p*K*a of PFOA in water, in order to better understand their environmental fate. The p*K*a values of PFCs at 25°C are summarized in Table 5.

Air-Water Partition Constants (K_{aw}) or Henry's Law Constant (HLC)

The air–water partition coefficient, expressed either in dimensionless form as K_{aw} or with units as the Henry's law constant (HLC), is useful to determine the equilibrium partition of a chemical between the air phase and the corresponding water phase (water bulk or atmospheric moisture). PFSAs are strong acids, thus they are expected to ionize and stay in the water bulk as anions. PFCAs are relatively weak acids that can partially exist in the nondissociated form depending on the pH value of the water phase. Reported values of K_{aw} for PFCAs and other weak-acid-like PFCs may thus

be influenced by the pKa of the chemicals and the pH values of the corresponding water phase. The K_{aw} of the pure compound is defined as the air–water partition coefficient of the fully associated (protonated) species, which is directly measured at pH values well below the pKa to ensure that virtually all of the molecules are protonated. The effective air–water partition coefficient ($K_{aw,\,eff}$) can be described as $K_{aw,\,eff} = K_{aw}° / (1 + 10^{pH-pKa})$ considering the effect of pH[84]. As there are some uncertainties for pKa, the derivation of $K_{aw,\,eff}$ from $K_{aw}°$ may pose some problems.

There are few experimental K_{aw} and/or HLC data for PFCAs. In fact, the K_{aw} values available for PFCAs are usually determined under sufficiently low pH conditions to suppress ionization of the corresponding chemicals. Bowden et al.[85] determined the HLC of TFA from measured partial pressures over aqueous solutions at 278.15, 298.15, and 308.15 K, and described $HLC_{aw}^{298.15}$ as a function of pKa (0.2~0.5) as

$$\mathrm{LnHLC}_{aw}^{298.15} = 8.282536 + 2.139651 \times pKa - 0.586898 \times pKa^{1.5}$$

The HLC at 298.15K of TFA was reported to be equal to 8950 ± 100 mol $kg^{-1}atm^{-1}$ with pKa = 0.47. The HLC values might have errors due to the uncertainties of pKa values. Using a similar procedure, Kutsuna and Hori[86] measured HLC values of TFA at 278.15, 288.15, and 298.15 K. Their values of the HLC (298.15K) were 0.65 times the reported value of Bowden et al.[85] for pKa = 0.47, and were equal to the value for pKa = 0.2. The authors pointed that the relatively small temperature dependence of HLC obtained in their research suggested that TFA may be transported from the water compartment at low pH into the atmosphere with less difficulty than previously thought. Li et al.[87] developed a modified gas-purge method to determine K_{aw}, which is suitable for substances that have low air-water partition coefficients and may aggregate in solution, ionize, and display surface activity. They determined the K_{aw} of PFOA as 1.02×10^{-3} at pH = 0.6 and a temperature of 20°C using this method. However, the accuracy of this method is dependent on the estimated efficiency of approach to equilibrium. These authors also mentioned that the pH of 0.6 used to necessitate complete protonation of PFOA in solution may have an impact on the measured K_{aw}.

Lei et al.[62] determined K_{aw} values of 4:2 FTOH, 6:2 FTOH, and 8:2 FTOH as a function of temperature using equilibrium static headspace GC and the phase ratio variation method. However, some errors may exist, as they used the GC-RT method with a nonpolar stationary phase, which did not fully account for the intermolecular interaction between target molecular and the water phase. Goss et al.[67] measured the K_{aw} of 4:2 FTOH, 6:2 FTOH, and 8:2 FTOH with static headspace measurements of solutions equilibrated with air at varying water/air ratios. The authors postulated that parallel lines are to be expected for the fluorinated alkanes and FTOHs that grow by -CF_2- increments in a logarithmic plot of K_{aw} versus the chain length. It is also

expected that classes of fluorinated compounds in which the chain length increases by one -CF_2- fragment should reveal a steeper slope than similar nonfluorinated compound classes as the -CF_2- fragment is larger and at the same time exhibits the same van der Waals interactions as a -CH_2- fragment. Comparing data on fluorinated compounds with the data of alkanes, n-perfluoroalkanes and the data of Lei et al.[62] Goss et al.[67] found that the data of Lei et al.[62] did not agree with the trend. The result of 8:2 FTOH reported by Lei et al.[62] also raised some questions, which were attributed to substantial sorption of 8:2 FTOH to interfaces. In addition, Goss et al.[67] pointed out that Lei et al.[62] were not able to measure the K_{aw} of any of the fluorotelomer olefins (FTolefin) as the nonpolar nature of these olefins induces less adsorption to the polar interface and less partitioning into the water phase. Goss et al.[67] therefore predicted K_{aw} values of fluorotelomer olefins using the polyparameter linear free energy relationships. Arp et al.[68] compared the experimental and predicted K_{aw} values of FTOHs at 25°C, and found that SPARC underestimates by about one order of magnitude, EPI Suite methods overestimate by up to four orders of magnitude, and COSMOtherm performed well only for 4:2 FTOH and 6:2 FTOH.

Table 6 provides an overview of measured K_{aw} values, whereas an overview of calculated values is shown in Table 7.

Lipophilicity and Octanol/Water Partition Coefficient (K_{ow})

The octanol/water partition coefficient (K_{ow}) is the ratio of the concentrations of a chemical in the n-octanol phase and in the water phase at equilibrium at a specified temperature. As n-octanol is used as a surrogate for fat or natural organic matter, K_{ow} can be used to represent the tendency of a chemical to partition between an organic phase (e.g., fish tissue or soil) and an aqueous phase. K_{ow} is thus a measure of the extent of lipophilicity or hydrophobicity of a chemical. As such, K_{ow} is usually related with soil/sediment adsorption coefficients, bioconcentration factors, and toxicity, and is a key parameter in the assessment of the environmental fate of organic chemicals.

As many PFCs are hydrophobic and lipophobic at the same time, they tend to form three immiscible layers when they are added to an octanol-water system. Thus, it is impossible to directly determine their K_{ow} values using 'regular' methods that are common for organic chemicals. Experimental K_{ow} data for PFCs are therefore very scarce. Furthermore, PFCs are fully or partially ionizable in water at conditions that are representative for the environment. Therefore, two kinds of octanol/water partition coefficients are used to quantify the partitioning of PFCs: one for the neutral species and the other for the ionized molecule.[88] In general, P indicates the partitioning of neutral species between octanol and water, while D refers to the total partition of a chemical (the neutral molecule and ionized species) between

TABLE 6. Overview of experimentally derived K_{aw} values of selected PFCs

Chemical	K_{aw}/HLC	Temperature (K)	pH	Method	Reference
TFA	$8.95 \times 10^{3} molkg^{-1}atm^{-1}$	298.15	0.47	partial pressures over aqueous solutions	85
TFA	$5.8 \times 10^{3} moldm^{-3}atm^{-1}$	298.15	0.47	partial pressures over aqueous solutions	86
4:2 FTOH	$1.74 \times 10^{2} Pam^{3}mol^{-1}$	308.15		Headspace GC	62
	$2.58 \times 10^{2} Pam^{3}mol^{-1}$	318.15		Headspace GC	62
	$3.69 \times 10^{2} Pam^{3}mol^{-1}$	328.15		Headspace GC	62
	$4.89 \times 10^{2} Pam^{3}mol^{-1}$	338.15		Headspace GC	62
	$6.18 \times 10^{2} Pam^{3}mol^{-1}$	348.15		Headspace GC	62
	$8.56 \times 10^{2} Pam^{3}mol^{-1}$	358.15		Headspace GC	62
6:2 FTOH	$2.40 \times 10^{2} Pam^{3}mol^{-1}$	318.15		Headspace GC	62
	$4.13 \times 10^{2} Pam^{3}mol^{-1}$	328.15		Headspace GC	62
	$6.71 \times 10^{2} Pam^{3}mol^{-1}$	338.15		Headspace GC	62
	$1.02 \times 10^{3} Pam^{3}mol^{-1}$	348.15		Headspace GC	62
	$1.49 \times 10^{3} Pam^{3}mol^{-1}$	358.15		Headspace GC	62
8:2 FTOH	$6.5 \times 10^{2} Pam^{3}mol^{-1}$	338.15		Headspace GC	62
	$1.16 \times 10^{3} Pam^{3}mol^{-1}$	348.15		Headspace GC	62
	$2.06 \times 10^{3} Pam^{3}mol^{-1}$	358.15		Headspace GC	62
	$2.83 \times 10^{2} Pam^{3}mol^{-1}$	363.15		Headspace GC	62
4:2 FTOH	$3.0 \times 10^{-2} m^{3}/m^{3}$	298.15		static headspace measurements	67
6:2 FTOH	$2.8 \times 10^{-1} m^{3}/m^{3}$	298.15		static headspace measurements	67
8:2 FTOH	$3.80\ m^{3}/m^{3}$	298.15		static headspace measurements	67
PFOA	1.02×10^{-3}	293.15	0.6	a modified gas-purge method	87

TABLE 7. Predicted K_{aw} values of selected PFCs at 25°C

Chemical	Kaw	Method	Reference
4:2 FTOH	1.23×10^2 Pam^{3}mol^{-1}	Extrapolated	62
6:2 FTOH	8.32×10Pam^3 mol^{-1}	Extrapolated	62
8:2 FTOH	3.72×10Pam^3 mol^{-1}	Extrapolated	62
4:2 FTOH	5.5×10^{-2} m^3/m^3	Calculated	67
6:2 FTOH	2.1×10^{-1}m^3/m^3	Calculated	67
8:2 FTOH	1 m^3/m^3	Calculated	67
10:2 FTOH	3.63 m^3/m^3	Calculated	67
4:2 FTolefin	1.58×10^2 m^3/m^3	Calculated	67
6:2 FTolefin	7.41×10^2m^3/m^3	Calculated	67
8:2 FTolefin	2.82×10^3 m^3/m^3	Calculated	67
10:2 FTolefin	1.17×10^4 m^3/m^3	Calculated	67
4:2 FTolefin	1.07×10^2(molL^{-1})/(molL^{-1})	Calculated	68
6:2 FTolefin	3.98×10^2(molL^{-1})/(molL^{-1})	Calculated	68
8:2 FTolefin	1.78×10^3(molL^{-1})/(molL^{-1})	Calculated	68
10:2 FTolefin	7.24×10^3(molL^{-1})/(molL^{-1})	Calculated	68
12:2 FTolefin	3.47×10^4(molL^{-1})/(molL^{-1})	Calculated	68
4:2 FTOH	3.2×10^{-2}(molL^{-1})/(molL^{-1})	Calculated	68
6:2 FTOH	1.9×10^{-1}(molL^{-1})/(molL^{-1})	Calculated	68
8:2 FTOH	5.4×10^{-1}(molL^{-1})/($molL^{-1}$)	Calculated	68
10:2 FTOH	4.57 (mol L^{-1})/(mol L^{-1})	Calculated	68
N-EtFOSA	6.3×10^{-2}(molL^{-1})/(molL^{-1})	Calculated	68
N-MeFOSE	8.3×10^{-4}(molL^{-1})/(molL^{-1})	Calculated	68
N-EtFOSE	7.1×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
N-MeFOSEA	9.1×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
PFHxA	9.1×10^{-4}(molL^{-1})/(molL^{-1})	Calculated	68
PHFpA	2.2×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
PFOA	4.3×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
PFNA	9.3×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
PFDA	1.6×10^{-2}(molL^{-1})/(molL^{-1})	Calculated	68
PFUnA	3.0×10^{-2}(molL^{-1})/(molL^{-1})	Calculated	68
PFDoA	8.7×10^{-2}(molL^{-1})/(molL^{-1})	Calculated	68
PFOS	4.0×10^{-3}(molL^{-1})/(molL^{-1})	Calculated	68
PFOSA	1.2×10^{-4}(molL^{-1})/(molL^{-1})	Calculated	68

two phases. As the ionized proportion of fluorinated weak acids is pH-dependent, their effective K_{ow} values are also pH dependent. Some software, such as SPARC (University of Georgia, Athens, GA, USA), ADMET Predictor™ (Simulations Plus, Inc., Lancaster, CA, USA), and Bio-Loom (BioByte Corp., Claremont, CA, USA), can be used to estimate both log P and log D [3,88].

Arp et al.[68] calculated "dry" K_{ow} values from experimental K_{aw} and K_{oa} data for 4:2 FTOH, 6:2 FTOH, and 8:2 FTOH, but they noted that the "dry" K_{ow} values are usually higher than "wet" estimates by a factor of 2~3. The difference between the dry K_{ow} and the wet K_{ow} is that the former does not take account of water molecules in the octanol phase and octanol molecules in the water phase at phase equilibrium.[89] In the supporting information, Arp et al.[68] provided K_{ow} values for other PFCs as well. Carmosini and Lee[90] measured logK_{ow} values of 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH using an equilibration method. The data reported are in relatively

TABLE 8. Overview of experimental logK_{ow} values of selected PFCs

Chemical	logK_{ow}	Method	Reference
PFBA	−0.52	ion-transfer cyclic voltammetry	91
PFPeA	0.09	ion-transfer cyclic voltammetry	91
PFHxA	0.70	ion-transfer cyclic voltammetry	91
PFHpA	1.31	ion-transfer cyclic voltammetry	91
PFOA	1.92	ion-transfer cyclic voltammetry	91
PFNA	2.57	ion-transfer cyclic voltammetry	91
PFDA	2.90	ion-transfer cyclic voltammetry	91
PFOS	2.45	ion-transfer cyclic voltammetry	91
$CF_3(CF_2)_5(CH_2)_2CO_2$–	−0.05	ion-transfer cyclic voltammetry	91
4:2 FTOH	3.30	an equilibration method	90
6:2 FTOH	4.54	an equilibration method	90
8:2 FTOH	5.58	an equilibration method	90
10:2 FTOH	6.63	an equilibration method	90

good agreement with estimates from Arp et al.[68] However, their K_{ow}values for the longer chain FTOHs diverged from those calculated by Arp et al.[68] Carmosini and Lee[90] pointed out that the deviation for the longer chain FTOHs is because of the difference of wet and dry logK_{ow}.

Jing et al.[91] determined octanol/water partition coefficients of a homologous series of perfluoroalkyl and alkyl carboxylates using ion-transfer cyclic voltammetry. It was found that perfluoroalkyl carboxylates are ~2 orders of magnitude more lipophilic than the corresponding alkyl carboxylates with the same carbon chain length, which is ascribed to the oxoanion group. The strong electron-withdrawing effect of the perfluoroalkyl group on the adjacent oxoanion group leads to weak hydration, and then decreases its hydrophilicity. Tables 8 and 9 provide overviews of reported values of log K_{ow} for PFCs.

Octanol/Air Partition Coefficient (K_{oa})

The octanol/air partition coefficient (K_{oa}) is a key property of organic chemicals to assess their partitioning between the atmosphere and organic phases such as organic films on aerosols, organic carbon in soil, the waxy cuticle, or lipid portions of vegetation. Determination of K_{oa} values focuses on PFCs that are easily transported into the atmosphere and likely to stay in this compartment, such as FTOHs.

Shoeib et al.[63] measured K_{oa} values of N-MeFOSE, N-EtFOSE, and N-MeFOSEA using a modified generator column method at 0, 10, and 20°C. The results showed that the logK_{oa} values of the chemicals tested increased with reciprocal absolute temperature, which means that the chemicals prefer to partition to organic phases at colder temperatures.

Lei et al.[62] estimated K_{oa} as a function of temperature for FTOHs and some fluorinated aromatics based on gas chromatographic retention times on

TABLE 9. Overview of calculated logK_{ow} values of selected PFCs at 25°C

Chemical	logK_{ow}	Method	Reference
4:2 Olefin	3.3	Calculated	68
6:2 Olefin	4.35	Calculated	68
8:2 Olefin	5.4	Calculated	68
10:2 Olefin	6.44	Calculated	68
12:2 Olefin	7.52	Calculated	68
4:2 FTOH	2.31	Calculated	68
6:2 FTOH	3.32	Calculated	68
8:2 FTOH	4.31	Calculated	68
10:2 FTOH	5.39	Calculated	68
N-EtFOSA	5.49	Calculated	68
N-MeFOSE	4.8	Calculated	68
N-EtFOSE	5.39	Calculated	68
N-MeFOSEA	6.25	Calculated	68
PFHxA	3.26	Calculated	68
PFHpA	3.82	Calculated	68
PFOA	4.3	Calculated	68
PFNA	4.84	Calculated	68
PFDA	5.3	Calculated	68
PFUnA	5.76	Calculated	68
PFDoA	6.41	Calculated	68

a nonpolar DB-1 capillary column. The data reported maybe underestimate the actual values because of the use of nonpolar stationary phase. Goss et al.,[67] determined K_{oa} of FTOHs using the method described by Shoeib et al.,[63] and found that their data exceed the data of Lei et al.[62] by more than one order of magnitude. Goss et al.[67] pointed out that octanol can principally not be mimicked by a DB-1 phase and the GC-retention data are expected to underestimate the actual logK_{oa} values. Arp et al.[68] compared the experimental and predicted K_{oa} of selected PFCs, evaluated the predictive performance of three software packages, and provided calculation results for some PFCs. Thuens et al.[92] determined K_{oa} values for five FTOHs (4:2 FTOH, 6:2 FTOH, 8:2 FTOH, 10:2 FTOH, and 12:2 FTOH) over the temperature range of 5–40°C using a modified generator column method. Their results agree with those published by Goss et al.[67] but do not agree with those of Lei et al.[62]

Dreyer et al.[93] determined the K_{oa} of three fluorotelomer acrylates (6:2 FTA, 8:2 FTA, 10:2 FTA), two perfluoroalkyl sulfonamides (N-MeFOSA, N-EtFOSA), and two perfluoroalkyl sulfonamido ethanols (N-MeFOSE, N-EtFOSE) over the temperature range 278–313 K using a modified generator column method. Calibrated logK_{oa} values at 298 K ranged from 4.5 (6:2 FTA) to 6.7 (N-EtFOSE). LogK_{oa} values decreased with increasing temperature and increased in the order of 6:2 FTA, 8:2 FTA, 10:2 FTA, N-MeFOSA, N-MeFOSE, N-EtFOSA, and N-EtFOSE. Tables 10 and 11 summarize the logK_{oa} values of selected PFCs.

TABLE 10. Overview of experimental $\log K_{oa}$ values of selected PFCs

Chemical	Temperature (K)	$\log K_{oa}$	Method	Reference
4:2 FTOH	283.15	5.06	a generator column method	67
	283.15	5.02	a generator column method	67
	293.15	4.91	a generator column method	67
	293.15	4.76	a generator column method	67
	293.15	4.93	a generator column method	67
	298.15	3.26	GC retention time	62
	278.15	5.29	a generator column method	92
	283.15	5.15	a generator column method	92
	288.15	4.89	a generator column method	92
	293.15	4.65	a generator column method	92
	298.15	4.54	a generator column method	92
	303.15	4.45	a generator column method	92
	313.15	4.10	a generator column method	92
6:2 FTOH	273.15	6.26	a generator column method	67
	283.15	5.52	a generator column method	67
	283.15	5.85	a generator column method	67
	293.15	5.49	a generator column method	67
	293.15	5.46	a generator column method	67
	293.15	5.51	a generator column method	67
	298.15	3.56	GC retention time	62
	278.15	5.59	a generator column method	92
	283.15	5.39	a generator column method	92
	288.15	5.12	a generator column method	92
	293.15	4.91	a generator column method	92
	298.15	4.79	a generator column method	92
	303.15	4.68	a generator column method	92
	313.15	4.45	a generator column method	92
8:2 FTOH	273.15	6.72	a generator column method	67
	283.15	6.27	a generator column method	67
	283.15	6.13	a generator column method	67
	293.15	5.79	a generator column method	67
	293.15	5.80	a generator column method	67
	293.15	5.75	a generator column method	67
	298.15	4.17	GC retention time	62
	278.15	6.34	a generator column method	92
	283.15	6.05	a generator column method	92
	288.15	5.89	a generator column method	92
	293.15	5.65	a generator column method	92
	298.15	5.55	a generator column method	92
	303.15	5.45	a generator column method	92
	313.15	5.17	a generator column method	92
10:2 FTOH	298.15	4.83	GC retention time	62
	278.15	6.31	a generator column method	92
	283.15	6.27	a generator column method	92
	288.15	6.02	a generator column method	92
	293.15	5.83	a generator column method	92
	298.15	5.72	a generator column method	92
	303.15	5.59	a generator column method	92
	308.15	5.37	a generator column method	92
	313.15	5.26	a generator column method	92
12:2 FTOH	278.15	6.79	a generator column method	92
	283.15	6.57	a generator column method	92

(Continued on next page)

TABLE 10. Overview of experimental logK_{oa} values of selected PFCs *(Continued)*

Chemical	Temperature (K)	logK_{oa}	Method	Reference
	288.15	6.42	a generator column method	92
	293.15	6.39	a generator column method	92
	298.15	6.16	a generator column method	92
	303.15	6.06	a generator column method	92
	308.15	5.91	a generator column method	92
	313.15	5.89	a generator column method	92
Pentafluorotoluene	298.15	3.19	GC retention time	62
Pentafluorophenol	298.15	4.42	GC retention time	62
1,3,5-trichloro-2,4,6-trifluorobenzene	298.15	4.71	GC retention time	62
Octafluoronaphthalene	298.15	4.70	GC retention time	62
Decafluorobiphenyl	298.15	4.97	GC retention time	62
6:2 FTA	278	5.02	a generator column method	93
	283	4.81	a generator column method	93
	288	4.71	a generator column method	93
	293	4.72	a generator column method	93
	298	4.67	a generator column method	93
	303	4.62	a generator column method	93
	308	4.04	a generator column method	93
	313	3.99	a generator column method	93
8:2 FTA	278	5.90	a generator column method	93
	283	5.65	a generator column method	93
	288	5.52	a generator column method	93
	293	5.43	a generator column method	93
	298	5.34	a generator column method	93
	303	5.40	a generator column method	93
	308	4.80	a generator column method	93
	313	4.61	a generator column method	93
10:2 FTA	278	6.54	a generator column method	93
	283	6.32	a generator column method	93
	288	6.19	a generator column method	93
	293	5.78	a generator column method	93
	298	5.68	a generator column method	93
	303	5.68	a generator column method	93
	308	5.29	a generator column method	93
	313	5.08	a generator column method	93
N-MeFOSA	278	6.68	a generator column method	93
	283	6.62	a generator column method	93
	288	6.53	a generator column method	93
	293	6.44	a generator column method	93
	298	6.32	a generator column method	93
	303	6.24	a generator column method	93
	308	6.14	a generator column method	93
N-MeFOSE	278	6.69	a generator column method	93
	283	6.69	a generator column method	93
	288	6.64	a generator column method	93
	293	6.52	a generator column method	93
	298	6.48	a generator column method	93
	303	6.36	a generator column method	93
	308	6.26	a generator column method	93
	293.15	7.70	a generator column method	63
	298.15	6.78	GC retention time	62

TABLE 10. Overview of experimental $\log K_{oa}$ values of selected PFCs *(Continued)*

Chemical	Temperature (K)	$\log K_{oa}$	Method	Reference
N-EtFOSA	278	6.88	a generator column method	93
	283	6.89	a generator column method	93
	288	6.76	a generator column method	93
	293	6.72	a generator column method	93
	298	6.65	a generator column method	93
	303	6.54	a generator column method	93
	308	6.40	a generator column method	93
	298.15	5.86	GC retention time	62
N-EtFOSE	278	6.95	a generator column method	93
	283	6.94	a generator column method	93
	288	6.87	a generator column method	93
	293	6.75	a generator column method	93
	298	6.71	a generator column method	93
	303	6.59	a generator column method	93
	308	6.52	a generator column method	93
	293.15	7.78	a generator column method	63
	298.15	7.09	GC retention time	62
N-MeFOSEA	293.15	7.87	a generator column method	63

Organic Carbon/Water Partition Coefficient (K_{oc})

Given the hydrophobicity and oleophobicity of the perfluorinated chain and the hydrophilicity of the sulfonate or carboxylate head groups, it is likely that both hydrophobic and electrostatic effects influence anionic PFC sorption.[94] PFCs may therefore be absorbed into organic matter and/or adsorbed to the surface of soil/sediments during the sorption process. As the organic carbon matter content of soil/sediments varies greatly, the soil/sediment-water partition coefficient (K_d) is usually divided by the total organic carbon content to obtain the organic carbon/water partition coefficient (K_{oc}). K_{oc} can be used to compare and evaluate the distribution of organic chemicals between the organic phase of soil/sediment and the water phase.

Higgins and Luthy[94] evaluated the sorptive potential of three classes of PFCs, perfluorocarboxylates, perfluorosulfonates, and perfluorooctyl sulfonamide acetic acids, for five kinds of freshwater sediments. It was found that the sorption of these compounds was influenced by sediment-, solution-, and chemical-specific parameters. Sediment organic carbon, rather than sediment iron oxide content, was the dominant sediment-parameter affecting sorption, indicating the importance of hydrophobic interactions. Sorption also increased with increasing aqueous Ca^{2+} concentration and decreasing pH, suggesting that electrostatic interactions play a role. The perfluorocarbon chain length was the dominant structural feature influencing the sorption, with each CF_2 moiety contributing 0.50~0.60 log

TABLE 11. Overview of predicted logK_{oa} values of selected PFCs at 25°C

Chemical	logK_{oa}	Method	Reference
6:2 FTA	4.4	Calculated	93
8:2 FTA	5.2	Calculated	93
10:2 FTA	5.7	Calculated	93
N-MeFOSA	6.3	Calculated	93
N-MeFOSE	6.4	Calculated	93
N-EtFOSA	6.6	Calculated	93
N-EtFOSE	6.7	Calculated	93
4:2 FTOH	4.80	Extrapolated	67
6:2 FTOH	5.26	Extrapolated	67
8:2 FTOH	5.56	Extrapolated	67
4:2 Olefin	1.4	Calculated	68
6:2 Olefin	1.92	Calculated	68
8:2 Olefin	2.35	Calculated	68
10:2 Olefin	2.78	Calculated	68
12:2 Olefin	3.21	Calculated	68
4:2 FTOH	3.85	Calculated	68
6:2 FTOH	4.11	Calculated	68
8:2 FTOH	4.67	Calculated	68
4:2 FTOH	4.84	Calculated	68
N-EtFOSA	6.87	Calculated	68
N-MeFOSE	7.97	Calculated	68
N-EtFOSE	7.64	Calculated	68
N-MeFOSEA	8.43	Calculated	68
PFHxA	6.4	Calculated	68
PFHpA	6.6	Calculated	68
PFOA	6.8	Calculated	68
PFNA	7.01	Calculated	68
PFDA	7.24	Calculated	68
PFUnA	7.44	Calculated	68
PFDoA	7.65	Calculated	68
PFOS	7.8	Calculated	68
APFO	8.43	Calculated	68

units to the measured distribution coefficients. The sulfonate moiety contributed an additional 0.23 log units to the measured distribution coefficient, when compared to carboxylate analogs. In addition, the perfluorooctyl sulfonamide acetic acids demonstrated substantially stronger sorption than PFOS.

Liu and Lee[46] measured the sorption of 8:2 FTOH by five soils from water and cosolvent/water solution at 22.3°C. All sorption isotherms were well fitted by the linear sorption model. Sorption appeared to be driven by hydrophobic partitioning with an average logK_{oc} value of 4.13. The authors then investigated the effect of the fluorotelomer alcohol chain length on soil sorption using the same procedure.[47] It was found that soil organic carbon content (OC) was the key soil property influencing sorption of FTOHs and the prefluorocarbon chain length was the dominant structural feature for the sorption. Each -CF_2- moiety increased OC-normalized sorption coefficients

TABLE 12. Overview of experimental logK_{oc} values of selected PFCs

Chemical	Temperature (K)	logK_{oc}	Method	Reference
PFOA	—	2.11	Aqueous loss method	94
PFNA	—	2.50	Aqueous loss method	94
PFDA	—	2.92	Aqueous loss method	94
PFUnA	—	3.47	Aqueous loss method	94
PFOS	—	2.68	Aqueous loss method	94
PFDS	—	3.66	Aqueous loss method	94
N-MeFOSAA	—	3.35	Aqueous loss method	94
N-EtFOSAA	—	3.49	Aqueous loss method	94
4:2 FTOH	295.65	0.933	Cosolvent extrapolated	47
6:2 FTOH	295.65	2.43	Cosolvent extrapolated	47
8:2 FTOH	295.45	4.13	Cosolvent extrapolated	46
10:2 FTOH	295.65	6.20	Cosolvent extrapolated	47

(K_{oc}) by ~0.87 log units, which is larger than that of ~0.6 log units predicted by Goss et al.[67] and that of 0.5–0.6 log units reported by Higgins and Luthy.[94] Table 12 provides an overview of measured values of logK_{oc}.

Bioaccumulation

Organisms accumulate high concentrations of certain organic contaminants relative to concentrations of these substances in the environment they inhabit, which may be referred to as the bioaccumulation potential.[95,96] The bioaccumulation potential can be characterized as bioconcentration factor (BCF), bioaccumulation factor (BAF), or biomagnification factor (BMF). BCF is defined as the ratio of the chemical concentration in an organism to the total chemical concentration in the corresponding water, and is usually determined under laboratory conditions. BAF expresses the uptake of a chemical from multiple exposure routes such as water and food, and can be estimated from laboratory experiments or data collected in the field. BMF is the ratio of the concentration of a chemical in an organism to that in the organism's diet. BMF can therefore be regarded as a special case of bioaccumulation in which the chemical concentration in the organism exceeds that in the organism's diet due to dietary absorption.

As PFCs are expected to reside in water, their bioaccumulation in aquatic organisms has been intensively investigated. Because of their hydrophobic and lipophobic nature, they are not accumulated in lipids of organisms as other POPs. Several studies have shown that they tend to bind on/to proteins, so protein-rich tissues such as liver, kidney and blood are their main repositories, with concentrations that are orders of magnitude higher than concentrations in other biological compartments.[28–31,97–99]

Martin et al.[30] investigated the dietary accumulation of a homologous series of PFCAs and PFSAs in juvenile rainbow trout (*Oncorhynchus mykiss*)

by exposure via the diet during 34 days, followed by a 41-day depuration period. BAFs were found to range from 0.038 to 1.0 and increased with the length of the perfluorinated chain. PFSAs bioaccumulate to a greater extent than PFCAs with equivalent perfluoroalkyl chain length, which shows that the acid functional group also affects the bioaccumulation potential. However, the results indicated that BAFs were not statistically greater than 1 for any PFAA, suggesting that dietary exposure will not result in biomagnification of PFAAs in juvenile trout. Subsequently, the authors tested the compound-specific tissue distribution and BCFs of these PFAAs using a flow-through exposure system for 12 days, followed by 33 days of depuration in clean water.[31] The results showed that PFCAs and PFSAs with perfluoroalkyl chain lengths shorter than seven and six carbon atoms, respectively, could not be detected in most tissues and were considered to have insignificant BCFs. For detectable PFAAs, carcass BCFs increased with increasing length of the perfluoroalkyl chain, ranging from 4.0 to 23,000, based on wet weight concentrations. Carboxylate carcass BCFs increased by a factor of eight for each additional carbon in the perfluoroalkyl chain between 8 and 12 carbons, but this relationship deviated from linearity for the longest PFAA tested, possibly because of decreased gill permeability. As for BAFs, PFSAs have greater BCFs than the corresponding PFCAs of equal perfluoroalkyl chain length.

Taniyasu et al.[100] examined the concentrations and distribution of PFOS in samples of surface water, fish and bird blood and livers, and human blood collected in Japan. Notable concentrations of PFOS were found in surface water and fish from Tokyo Bay. Based on these data, BCFs for PFOS in livers of fishes were calculated as being in the range of 274–41600 (mean = 8540). The corresponding log BCF values in fishes were 2.4–4.6 (mean = 3.9). Ankley et al.[101] exposed the northern leopard frog (*Rana pipiens*) to PFOSK from early development through metamorphosis (54 days). They characterized the kinetics of PFOS bioconcentration in tadpoles using a one-compartment model. The BCFs obtained for the northern leopard frog were 83.1 and 27.7, respectively after 54 days of exposure to 0.1 mg L^{-1} and 1.0 mg L^{-1} PFOSK. It is interesting to see that BCF values for higher concentration PFOSK are lower than those determined at lower concentrations. Compared to juvenile rainbow trout, *Rana pipiens* was found to have a lower bioaccumulation potential.

Kannan et al.[102] investigated trophic transfer of some PFCs in a Great Lakes benthic foodweb. It was found that the BCF of PFOS is approximately 1000 for lower trophic-level benthic invertebrates, such as benthic algae, amphipods, and zebra mussels. Based on a whole-body PFOS concentration in round gobies, they estimated its BCF value to be 2400. As PFOA was not detected in benthic organisms, the chemical was shown to have a low bioaccumulation potential. PFOS tends to biomagnify in higher trophic level fishes such as salmonids. A BMF of 10~20 was determined between round

gobies and Chinook salmon liver, and a BMF of 5~10 was observed between salmon liver and eagle/mink livers.

Sinclair et al.[103] measured PFOS and PFOA in fish and birds from New York State. A BCF of 8850 was reported on the ratio of the concentration in the livers of fish and the surface water for PFOS and an average BMF of 8.9 for common mergansers to fish. For PFOA, a BCF of 184 was estimated for the livers of fish, while PFOA was not detected in the livers of any of the birds, which indicted a lower bioaccumulation potential of PFOA as compared to PFOS.

Morikawa et al.[104] measured BCFs of PFOS and PFOA in wild turtles (*Trachemys scripta elegans* and *Chinemys reevessii*). The geometric mean (geometric standard deviation) of BCFs of PFOS and PFOA were measured to be 10964 (2.5) and 3.2 (7.9), respectively. They found that the BCF of PFOA decreased significantly as the PFOA concentration increased, while there was no relationship between the BCF and the concentration of PFOS in surface water. Therefore, the authors suggested that absorption of PFOA in the gut might be a saturable process.

Houde et al.[105] investigated the biomagnification of a suite of PFCs in the food web of the bottlenose dolphin (*Tursiops truncatus*). They measured concentrations of selected PFCs in surficial seawater and sediment samples, as well as zooplankton, fish, and bottlenose dolphin tissue samples at Sarasota Bay (Florida) and Charleston Harbor (South Carolina) of the United States. It was found that BMFs ranged from <1 to 156 at Sarasota Bay and from <1 to 30 at Charleston. Based on the experimental data, the authors stressed that using plasma and liver PFC concentrations as surrogate to whole body burden in a top marine predator may overestimate the BMFs and TMFs.

Furdui et al.[106] investigated the spatial distribution of PFCs in lake trout from the Great Lakes using individual whole-body homogenates of 4-year-old lake trout (*Salvelinus namaycush*) samples. They obtained whole body BAFs by dividing the average concentration of PFCs in lake trout by the average concentration in water from each lake, which showed a remarkably narrow range of BAFs among lakes. For PFSAs the highest logBAFs were calculated for PFOS (4.1), followed by PFOSA (3.8) and PFHxS (2.7). BAFs for PFCAs showed a linear increase from PFOA (3.2) to PFDA (3.9), with PFDA having a similar BAF to PFOS and PFOSA.

Haukås et al.[107] reported the biomagnification potential of PFCs in species of the Barents Sea food web. The examined species included: sea ice amphipod (*Gammarus wilkitzkii*), polar cod (*Boreogadus saida*), black guillemot (*Cepphus grylle*), and glaucous gull (*Larus hyperboreus*). It was found that BMFs of PFHxS, PFOS, $\sum$PFAAS, and PFNA were >1, which implied biomagnification of these chemicals in the Barents Sea food web. Although accumulating through different pathways, the degree of trophic

transfer of PFAS is similar to that of the lipid soluble contaminants PCBs, DDTs, and PBDEs.

Higgins et al.[108] measured the biological uptake and elimination of selected PFCAs, PFSAs, and N-ethyl perfluorooctane sulfonamido acetic acid (N-EtFOSAA) in sediments by the freshwater oligochaete, *Lumbriculus variegatus*. The results showed that PFCs in sediments are readily bioavailable and that bioaccumulation from sediments does not continually increase with increasing perfluorocarbon chain length. This disagreement among congeners may be related with the difference of uptake and elimination of PFCs. Based on the biota sediment accumulation factors (BSAFs) from laboratory and field, PFOS and PFNA are the most bioaccumulative PFCs studied. It was found that N-EtFOSAA accumulated in the worm tissues and appeared to undergo biotransformation to PFOS, which may contribute to the bioaccumulation of PFOS.

Houde et al.[109] reported the fractionation and bioaccumulation of PFOS isomers in a Lake Ontario food web. The results showed that linear PFOS (L-PFOS) took a much higher proportion of total PFOS in all organisms, which may be related with a reduced uptake of branched isomers, a more rapid elimination of the branched isomers and/or a selective retention of the L-PFOS. The BAF of L-PFOS between lake trout (whole fish) and water was estimated to be 3.4×10^4 L kg^{-1} compared with 2.9×10^3 L kg^{-1} for the monomethyl-substituted group (MM-PFOS). A greater partitioning potency to biota and sediment of L-PFOS than branched isomers was ascribed to an enrichment of branched isomers in water.

Conder et al.[96] reviewed bioaccumulation potentials of PFCAs, and presented five conclusions.

1. Bioconcentration and bioaccumulation of perfluorinated acids are directly related to the length of each compound's fluorinated carbon chain.
2. PFSAs are more bioaccumulative than PFCAs of the same fluorinated carbon chain length.
3. PFCAs with seven fluorinated carbons or less (PFO and shorter PFCAs) are not considered bioaccumulative according to the range of promulgated bioaccumulation, "B", regulatory criteria of 1000–5000 L kg^{-1}.
4. PFCAs with seven fluorinated carbons or less have low biomagnification potential in food webs.
5. More research is necessary to fully characterize the bioaccumulation potential of PFCAs with longer fluorinated carbon chains (>7 fluorinated carbons) as PFCAs with longer fluorinated carbon chains may exhibit partitioning behavior similar to or greater than PFOS.

The reported bioaccumulation potential of PFCs is shown as follows: Table 13 for BCF values, Table 14 for BAF values, Table 15 for BSAFs values, and Table 16 for BMF values.

TABLE 13. BCF values of selected PFCs

Chemical	BCF	Organism	Organ	Reference
PFOA	4 ($L\ kg^{-1}$)	rainbow trout	Carass	31
PFDA	4.50×10^2 (Lkg^{-1})	rainbow trout	Carass	31
PFUnA	2.70×10^3(Lkg^{-1})	rainbow trout	Carass	31
PFDoA	1.80×10^4(Lkg^{-1})	rainbow trout	Carass	31
PFTeA	2.30×10^4(Lkg^{-1})	rainbow trout	Carass	31
PFOS	1.10×10^3 (Lkg^{-1})	rainbow trout	Carass	31
PFHxS	9.6 ($L\ kg^{-1}$)	rainbow trout	Carass	31
PFOA	2.7×10(Lkg^{-1})	rainbow trout	Blood	31
PFDA	2.70×10^3 (Lkg^{-1})	rainbow trout	Blood	31
PFUnA	1.10×10^4 (Lkg^{-1})	rainbow trout	Blood	31
PFDoA	4.0×10^4 (Lkg^{-1})	rainbow trout	Blood	31
PFTeA	3.0×10^4 (Lkg^{-1})	rainbow trout	Blood	31
PFOS	4.3×10^3 (Lkg^{-1})	rainbow trout	Blood	31
PFHxS	7.6×10($L\ kg^{-1}$)	rainbow trout	Blood	31
PFOA	8.0 ($L\ kg^{-1}$)	rainbow trout	Liver	31
PFDA	1.10×10^3 (Lkg^{-1})	rainbow trout	Liver	31
PFUnA	4.90×10^3 (Lkg^{-1})	rainbow trout	Liver	31
PFDoA	1.80×10^4 (Lkg^{-1})	rainbow trout	Liver	31
PFTeA	3.0×10^4 (Lkg^{-1})	rainbow trout	Liver	31
PFOS	5.40×10^3 (Lkg^{-1})	rainbow trout	Liver	31
PFHxS	1.0×10^2 (Lkg^{-1})	rainbow trout	Liver	31
PFOSK (0.1 mg L^{-1})	8.31×10	northern leopard frog	Whole body	101
PFOSK (1.0 mg L^{-1})	2.77×10	northern leopard frog	Whole body	101
PFOA	3.2	wild turtle	serum	104
PFOS	1.10×10^4	wild turtle	serum	104
PFOS	1.0×10^3	benthic invertebrates	Whole body	102
PFOS	2.40×10^3	Round Goby	Whole body	102
PFOS	8.85×10^3	Fish	Liver	103
PFOS	$2.74 \times 10^2 \sim 4.16 \times 10^4$	Fish	Liver	100
PFOA	1.84×10^2	Fish	Liver	103

AQUATIC TOXICITY

Aquatic Plants

In a document of OECD,[39] results of a seven-day growth inhibition test of PFOSK on *Lemna gibba* (Duckweed) were reported. The test yielded a seven-day IC_{50} of 108 mg L^{-1} for inhibition of frond production and a seven-day no observed effect concentration (NOEC) of 15.1 mg L^{-1} based on the inhibition of frond production, and evidence of sublethal effects. Boudreau et al.[110] also evaluated the toxicity of PFOSK on the floating macrophyte *Lemna gibba* using standard laboratory protocols. The seven-day IC_{50} was found to be 59.1 mg L^{-1} for frond number and 31.1 mg L^{-1} for wet weight. The NOECs from frond number and wet weight were 29.2 and 6.6 mg L^{-1}, respectively. Wet weight decreased along with a visual decrease in mean frond size, root length, and frond number over the concentrations of

TABLE 14. BAF values of selected PFCs

Chemical	BAF	Organism	Organ	Reference
PFOA	3.8×10^{-2}	rainbow trout	Carass	30
PFDA	2.3×10^{-1}	rainbow trout	Carass	30
PFUnA	2.8×10^{-1}	rainbow trout	Carass	30
PFDoA	4.3×10^{-1}	rainbow trout	Carass	30
PFTeA	1.0	rainbow trout	Carass	30
PFOS	$3.2 \times 10_{-1}$	rainbow trout	Carass	30
PFHxS	$1.4 \times 10_{-1}$	rainbow trout	Carass	30
L-PFOS	$6.5 \times 10^{2} L\,kg^{-1}$	zooplankton		109
	$3.0 \times 10^{3} L\,kg^{-1}$	Mysis		109
	$3.2 \times 10^{4} L\,kg^{-1}$	Diporeia		109
	$2.4 \times 10^{4} L\,kg^{-1}$	alewife		109
	$4.5 \times 10^{4} L\,kg^{-1}$	smelt		109
	$2.34 \times 10^{5} L\,kg^{-1}$	sculpin		109
	$3.4 \times 10^{4} L\,kg^{-1}$	lake trout		109
MM-PFOS	$1.7 \times 10^{2} L\,kg^{-1}$	Mysis		109
	$9.6 \times 10^{2} L\,kg^{-1}$	Diporeia		109
	$1.8 \times 10^{3} L\,kg^{-1}$	alewife		109
	$1.46 \times 10^{4} L\,kg^{-1}$	smelt		109
	$2.9 \times 10^{3} L\,kg^{-1}$	sculpin		109
	$1.7 \times 10^{2} L\,kg^{-1}$	lake trout		109
PFHxS	5.01×10^{2}	lake trout	Whole body	106
PFOS	1.26×10^{4}	lake trout	Whole body	106
PFOSA	6.31×10^{3}	lake trout	Whole body	106
PFOA	1.58×10^{3}	lake trout	Whole body	106
PFNA	3.98×10^{3}	lake trout	Whole body	106
PFDA	7.94×10^{3}	lake trout	Whole body	106

40 mg L^{-1}. Plants exposed to the highest concentration of 160 mg L^{-1} exhibited a high percentage of chlorosis as well as necrosis. Compared with green algae *Selenastrum capricornutum* and *Chlorella vulgaris*, *L. gibba* is more sentitive to PFOSK based on the inhibition of growth.

Hanson et al.[111] evaluated the toxicity of PFOSK to the aquatic macrophytes *Myriophyllum sibiricum* and *M. spicatum* under seminatural field conditions using 12000 L outdoor microcosms. *M. sibiricum* was more sensitive to PFOSK under these simulated field conditions than *M. spicatum*. The

TABLE 15. Steady-state BSAFs of selected PFCs

Chemical	BSAF	Organism	Reference
PFOA	33 ± 12	Lumbriculus variegatus	108
PFNA	55 ± 24	*Lumbriculus variegatus*	108
PFDA	35 ± 15	*Lumbriculus variegatus*	108
PFUnA	21 ± 9	*Lumbriculus variegatus*	108
PFDoA	19 ± 7	*Lumbriculus variegatus*	108
PFOS	42 ± 17	*Lumbriculus variegatus*	108
PFDS	17 ± 6	*Lumbriculus variegatus*	108
N-EtFOSAA	7 ± 2	*Lumbriculus variegatus*	108

TABLE 16. BMF values of selected PFCs

Chemical	BMF	Organism	Location	Reference
PFOA	7.2	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	13	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	13	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	2.7	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	2.3	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	6.4	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	1.8	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFNA	1.5	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	5	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	3.2	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	1.4	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	24	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	4.6	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	2.1	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFDA	3.7	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	2.9	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	8.8	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	2.4	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	2.5	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	2.8	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	2.4	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFUnA	0.9	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	1.9	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	2.4	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	3.2	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	2.1	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	3.9	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	2.5	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFDoA	0.1	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	0.2	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	0.1	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	0.4	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	1.8	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	0.6	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	0.6	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFHxS	nc	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	4	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	nc	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	14	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	nc	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	6	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	3.3	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFOSA	24	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	8.3	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	30	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105
	3.4	$dolphin_{whole}/red\ drum_{whole}$	Charleston, SC	105
	1.5	$dolphin_{whole}/Atlantic\ croaker_{whole}$	Charleston, SC	105
	4.4	$dolphin_{whole}/spotfish_{whole}$	Charleston, SC	105
	1.3	$dolphin_{whole}/seatrout_{whole}$	Charleston, SC	105
PFOS	4.6	$seatrout_{whole}/pinfish_{whole}$	Charleston, SC	105
	2.6	$dolphin_{whole}/striped\ mullet_{whole}$	Charleston, SC	105
	4	$dolphin_{whole}/pinfish_{whole}$	Charleston, SC	105

(Continued on next page)

TABLE 16. BMF values of selected PFCs *(Continued)*

Chemical	BMF	Organism	Location	Reference
	1.2	$dolphin_{whole}$/$red\ drum_{whole}$	Charleston, SC	105
	2.2	$dolphin_{whole}$/$Atlantic\ croaker_{whole}$	Charleston, SC	105
	0.8	$dolphin_{whole}$/$spotfish_{whole}$	Charleston, SC	105
	0.9	$dolphin_{whole}$/$seatrout_{whole}$	Charleston, SC	105
PFDoA	89	$striped\ mullet_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	2.5	$pigfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	156	$sheephead_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	2.5	$pinfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	35	$seatrout_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	0.4	$seatrout_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	14	$seatrout_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	0.2	$seatrout_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	14	$seatrout_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	0.1	$dolphin_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	2	$dolphin_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	0	$dolphin_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	2	$dolphin_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	0.1	$dolphin_{whole}$/$seatrout_{whole}$	Sarasota Bay, FL	105
PFHxS	nc	$striped\ mullet_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	9.1	$pigfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	nc	$sheephead_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	10	$pinfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	nc	$dolphin_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	2	$dolphin_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	nc	$dolphin_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	1.8	$dolphin_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	nc	$dolphin_{whole}$/$seatrout_{whole}$	Sarasota Bay, FL	105
PFOSA	2.5	$striped\ mullet_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	nc	$pigfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	2.5	$sheephead_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	nc	$pinfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	2.5	$seatrout_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	1	$seatrout_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	1	$seatrout_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	nc	$seatrout_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	5.2	$dolphin_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105
	nc	$dolphin_{whole}$/$pigfish_{whole}$	Sarasota Bay, FL	105
	5.2	$dolphin_{whole}$/$sheephead_{whole}$	Sarasota Bay, FL	105
	nc	$dolphin_{whole}$/$pinfish_{whole}$	Sarasota Bay, FL	105
	5.2	$dolphin_{whole}$/$seatrout_{whole}$	Sarasota Bay, FL	105
PFOS	23	$striped\ mullet_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	12	$pigfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	14	$sheephead_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	19	$pinfish_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	35	$seatrout_{whole}$/$zooplankton_{whole}$	Sarasota Bay, FL	105
	1.5	$seatrout_{whole}$/$striped\ mullet_{whole}$	Sarasota Bay, FL	105

TABLE 16. BMF values of selected PFCs *(Continued)*

Chemical	BMF	Organism	Location	Reference
	2.8	$seatrout_{whole}/pigfish_{whole}$	Sarasota Bay, FL	105
	2.6	$seatrout_{whole}/sheephead_{whole}$	Sarasota Bay, FL	105
	1.8	$seatrout_{whole}/pinfish_{whole}$	Sarasota Bay, FL	105
	9.6	$dolphin_{whole}/striped\ mullet_{whole}$	Sarasota Bay, FL	105
	18	$dolphin_{whole}/pigfish_{whole}$	Sarasota Bay, FL	105
	16	$dolphin_{whole}/sheephead_{whole}$	Sarasota Bay, FL	105
	11	$dolphin_{whole}/pinfish_{whole}$	Sarasota Bay, FL	105
	6.2	$dolphin_{whole}/seatrout_{whole}$	Sarasota Bay, FL	105
PFHxS	—	Polar cod/Ice amphipod	the Barents Sea	107
	—	Black guillemot/Ice amphipod	the Barents Sea	107
	6.00	Black guillemot/Polar cod	the Barents Sea	107
	7.20	Glaucous gull/Polar cod	the Barents Sea	107
	8.49	Glaucous gull/Black guillemot	the Barents Sea	107
	—	Black guillemot/Mixed diet	the Barents Sea	107
	—	Glaucous gull/Mixed diet	the Barents Sea	107
PFOS	0.32	Polar cod/Ice amphipod	the Barents Sea	107
	1.54	Black guillemot/Ice amphipod	the Barents Sea	107
	10.1	Black guillemot/Polar cod	the Barents Sea	107
	38.7	Glaucous gull/Polar cod	the Barents Sea	107
	27.0	Glaucous gull/Black guillemot	the Barents Sea	107
	5.66	Black guillemot/Mixed diet	the Barents Sea	107
	11.3	Glaucous gull/Mixed diet	the Barents Sea	107
PFNA	—	Polar cod/Ice amphipod	the Barents Sea	107
	—	Black guillemot/Ice amphipod	the Barents Sea	107
	8.76	Black guillemot/Polar cod	the Barents Sea	107
	11.6	Glaucous gull/Polar cod	the Barents Sea	107
	9.34	Glaucous gull/Black guillemot	the Barents Sea	107
	—	Black guillemot/Mixed diet	the Barents Sea	107
	—	Glaucous gull/Mixed diet	the Barents Sea	107
$\sum$PFAS	0.41	Polar cod/Ice amphipod	the Barents Sea	107
	1.01	Black guillemot/Ice amphipod	the Barents Sea	107
	5.25	Black guillemot/Polar cod	the Barents Sea	107
	16.9	Glaucous gull/Polar cod	the Barents Sea	107
	22.8	Glaucous gull/Black guillemot	the Barents Sea	107
	3.15	Black guillemot/Mixed diet	the Barents Sea	107
	7.72	Glaucous gull/Mixed diet	the Barents Sea	107
L-PFOS	60	lake trout/zooplankton	Lake Ontario	109
	12	lake trout/mysis	Lake Ontario	109
	1.2	lake trout/diporeia	Lake Ontario	109
	1.6	lake trout/alewife	Lake Ontario	109
	0.84	lake trout/smelt	Lake Ontario	109
	0.16	lake trout/sculpin	Lake Ontario	109
	9.0	sculpin/diporeia	Lake Ontario	109
MM-PFOS	—	lake trout/zooplankton	Lake Ontario	109
	17	lake trout/mysis	Lake Ontario	109
	3.0	lake trout/diporeia	Lake Ontario	109
	1.6	lake trout/alewife	Lake Ontario	109
	0.91	lake trout/smelt	Lake Ontario	109
	0.20	lake trout/sculpin	Lake Ontario	109
	15	sculpin/diporeia	Lake Ontario	109
PFOS	10~20	Chinook salmon/Round goby	Great Lakes	102
PFOS	5~10	Eagle-mink/salmon	Great Lakes	102
PFOS	8.9	Common mergansers/fish	New York state	103

Note. nc = not calculated.

researchers chose a number of endpoints—including growth (plant length), biomass (wet mass/dry mass), root number (primary roots from the plant stem), primary root lengths (total and longest), number of nodes, chlorophyll *a*/*b*, and carotenoid content—to characterize the toxicity. Toxicity was observed in the evaluated end points at >3 mg L^{-1} PFOSK for EC_{10}s and >12 mg L^{-1} PFOSK for EC_{50}s for *M. spicatum*, and in *M. sibiricum* at >0.1 mg L^{-1} for EC_{10}s and >1.6 mg L^{-1} for EC_{50}s. The NOEC for *M. spicatum* was consistently ≥11.4 mg L^{-1} PFOSK, whereas the NOEC for *M. sibiricum* was ≥0.3 mg L^{-1} PFOSK. However, the distinct difference in sensitivity between these two species exposed to PFOSK was not observed for the toxicity of PFOA applied as the sodium salt.[112] Toxicity after 14–35 days of exposure in the evaluated endpoints for *M. spicatum* was ≥5.7 mg L^{-1} PFOA for EC_{10} and ≥31.8 mg L^{-1} PFOA for EC_{50}; in *M. sibiricum* this was ≥8.4 mg L^{-1} PFOA for EC_{10} and ≥35.8 mg L^{-1} PFOA for EC_{50}. The NOECs for *Myriophyllum spp.* were consistently ≥23.9 mg L^{-1} PFOA.

Phillips et al.[113] tested the seven-day acute toxicity of saturated and unsaturated fluorotelomer carboxylic acids (FTCAs and FTUCAs) on the floating macrophyte *Lemna gibba*. Endpoints were evaluated based on total frond number and dry biomass. Results revealed that toxicity increased with increasing fluorocarbon (FC) chain length, for 4–8 FCs, and the saturated forms of the telomer acids were usually more toxic than their unsaturated counterparts. Compared with *Daphnia magna* and *Chironomus tentans*, *L. gibba* was more sensitive to telomer acids of chain lengths ≤8 FCs. In addition, the authors pointed out that the fluorotelomer carboxylic acids, as precursors of PFCAs, were more toxic than the PFCAs themselves.

Li[114] tested the toxicity of PFOSK and APFO on three plant species: lettuce (*Lactuca sativa*), cucumber (*Cucumis sativus*), and pakchoi (*Brassica rapa* chinensis). It was found that both PFOS and PFOA had no obvious adverse effect on seed germination for all three plant species. The five-day EC_{50} of root elongation was more sensitive than the LC_{50} of seed germination in this study and PFOSK was more toxic than APFO for all species tested. Based on EC_{10}, EC_{50}, and NOECs, the five-day root elongation sensitivity of test plants to both PFOS and PFOA was in the order of lettuce (*Lactuca sativa*) > pakchoi (*Brassica rapa* chinensis) > cucumber (*Cucumis sativus*). Li indicated that current PFOSK and APFO levels in freshwater may have no acute harmful ecological impact on the aquatic environment. An overview of toxicity data of PFCs on aquatic plants is given in Table 17.

Algae

The hazard and risk assessment of PFOS reported by the OECD[39] lists some toxicity data of PFOSK for the freshwater algae, *Pseudokirchneriella subcapitata*, *Anabaena flos-aquae* and *Navicula pelliculosa*, and the saltwater algae *Skeletonema costatum*. The endpoints assessed in the tests were the

TABLE 17. Aquatic toxicity of PFCs to plants with values of $LC_{50}/IC_{50}/EC_{50}$ and NOEC (mg L^{-1})

Chemical	$LC_{50}/IC_{50}/EC_{50}$	NOEC	Organism	Reference
PFOSK	108 (7 days FP)	15.1 (7 days FP)	*Lemna gibba*	39
PFOSK	200 (SG); 99 (RE)	200 (SG); 50 (RE)	*Lactuca sativa*	114
APFO	1734 (SG); 170 (RE)	1000 (SG); < 62.5 (RE)	*Lactuca sativa*	114
PFOSK	200 (SG); 130 (RE)	200 (SG); 50 (RE)	*Brassica rapa* chinensis	114
APFO	579 (SG); 278 (RE)	250 (SG); 125 (RE)	*Brassica rapa* chinensis	114
PFOSK	200 (SG); > 200 (RE)	200 (SG); > 200 (RE)	*Cucumis sativus*	114
APFO	2000 (SG); 1254 (RE)	2000 (SG); 250 (RE)	*Cucumis sativus*	114
PFOSK	59.1 (seven-day GI-FN); 31.1 (seven-day GI-WW)	29.2 (seven-day GI-FN); 6.6 (seven-day GI-WW)	*Lemna gibba*	110
PFOSK	24.5/22.8/19.9 (14/28/42 days LR)	11.4 (14/28/42 days LR)	*Myriophyllum spicatum*	111
PFOSK	4.4/10.2/1.6 (14/28/42 days LR)	2.9/0.3/0.3 (14/28/42 days LR)	*Myriophyllum sibiricum*	111
PFOA Na	98.3/69.3/62.7 (14/28/42 days LR)	23.9 (14/28/42 days LR)	*Myriophyllum spicatum*	112
PFOA Na	44.9/43.3/52.0 (14/28/42 days LR)	23.9 (14/28/42 days LR)	*Myriophyllum sibiricum*	112
4:2 FTUCA	6.64 (DW)/13.06(FN)		*Lemna gibba*	113
6:2 FTUCA	5.02 (DW)/9.20(FN)		*Lemna gibba*	113
8:2 FTUCA	0.71 (DW)/0.92(FN)		*Lemna gibba*	113
10:2 FTUCA	4.84 (DW)/ > 5.42(FN)		*Lemna gibba*	113
4:2 FTCA	9.39 (DW)/25.39(FN)		*Lemna gibba*	113
6:2 FTCA	1.29 (DW)/3.68(FN)		*Lemna gibba*	113
8:2 FTCA	1.36 (DW)/1.85(FN)		*Lemna gibba*	113
10:2 FTCA	4.30 (DW)/ > 4.30(FN)		*Lemna gibba*	113

Note. DW = dry weight; GI = growth Inhibition; FP = frond production; FN = frond number; LR = longest root (cm); RE = root elongation; SG = seed germination; WW = wet weight.

growth measured in terms of cell density, growth rate and/or the area under the growth curve over 96 hr. The 96-hr EC_{50} values for effects on growth rate and cell density of *Pseudokirchneriella subcapitata* were determined as 126 mg L^{-1} and 71 mg L^{-1}, respectively. The 96-hr NOECs for these two endpoints were all equal to 44 mg L^{-1}. The tests with two other species yielded 96-hr EC_{50} values of 176 mg L^{-1} (*A. flos-aquae*) and 305 mg L^{-1} (*N. pelliculosa*) and respective NOECs of 94 and 206 mg L^{-1}. The 96-hr growth inhibition test of PFOSK on *Skeletonema costatum* was unable to determine a definitive 96-hr EC_{50} value because no effects were determined at the highest dissolved PFOSK concentration (3.2 mg L^{-1}).

Boudreau et al.[110] tested the 96-hr toxicity of PFOSK on the green algae *Selenastrum capricornutum* and *Chlorella vulgaris* using standard laboratory protocols. It was found that PFOSK significantly inhibited the growth of *S. capricornutum* and *C. vulgaris* at concentrations $\geq$50 mg L^{-1}. The 96-hr IC_{50}s of *S. capricornutum* were 48.2 and 59.2 mg L^{-1} as determined on the basis of cell density and chlorophyll (*a*) concentration, respectively. *C. vulgaris* was slightly less sensitive than *S. capricornutum*, with IC_{50}s of 81.6 and 88.1 mg L^{-1} for cell density and chlorophyll (*a*), respectively. NOECs derived from the more sensitive endpoint, cell density, were 5.3 and 8.2 mg L^{-1} for *S. capricornutum* and *C. vulgaris*, respectively.

Colombo et al.[115] tested the freshwater aquatic toxicity of APFO on green algae, *Pseudokirchneriella subcapitata*, following OECD test guideline 201 and EU Commission Directive 92/69/EEC. Results showed that acute 48~96 hr LC/EC_{50} values were greater than 400 mg L^{-1}, and the lowest NOEC was 12.5 mg L^{-1} for 96-hr inhibition of the growth rate and biomass of the freshwater alga. Based on calculated effect levels, these authors concluded that nonionized ammonia potentially has a significant contribution to the observed toxicity of APFO.

Liu et al.[116] tested the toxicity of PFBS, PFHxS, PFOS, PFHxA, PFOA, PFDoA, and PFTeA on the freshwater green alga *Scenedesmus obliquus*. They found that PFOS, PFDoA, and PFTeA inhibited algal growth in a concentration-dependent manner while PFBS, PFHxA, and PFOA did not inhibit the algal growth within the range of concentrations tested. It was concluded that both carbon chain length and nature of the acid group influenced the toxicity of PFAAs: the toxicity increased with increasing carbon chain length for compounds belonging to the same class. An observed enhancement of the mitochondrial membrane potential (MMP) and cell membrane permeability in *S. obliquus* was stated to be caused by exposure to PFOS, PFOA, PFDoA, and PFTeA. The observed effective concentrations lie in the micromolar range, and the test compounds disrupted membrane properties at concentrations below those associated with algal growth inhibition. Therefore, the authors pointed out that the effect of PFAAs on membrane function of freshwater alga may cause a serious risk to single-celled algae that are exposed directly to pollutants in the environment. Based on these

findings, Liu et al[117] investigated the effects of interactions between PFOS and other compounds on the growth rate in *Scenedesmus obliquus*. Single application of PFOS showed no inhibition on the growth of *S. obliquus* below 40 mg L^{-1}, whereas PFOS acting with pentachlorophenol resulted in higher algal growth inhibition in comparison with pentachlorophenol alone. On the contrary, the algal growth inhibition of atrazine and diuron was depressed by PFOS. It was found that cell uptake of pentachlorophenol increased while that of atrazine and diuron was reduced in cells that had been exposed to PFOS. Therefore, Liu et al.[116] suggested that PFOS influences the cell uptake and toxicity of structurally different compounds in dissimilar manners and potentially increases the accessibility and toxicity of more hydrophobic compounds to algal cells.

Latała et al.[118] tested the 72-hr toxicity of PFHxA, PFHpA, PFOA, and PFNA to three representative marine algae in the Baltic Sea, the green alga *Chlorella vulgaris*, the diatom *Skeletonema marinoi*, and the blue-green alga *Geitlerinema amphibium*. The EC_{50} values obtained range from 0.28 mM (129.9 mg L^{-1}) to 12.84 mM (4032 mg L^{-1}). The blue-green alga and diatom were far more sensitive to PFCAs than the green alga, which was explained on the basis of differences in the cell wall structure. It was found that the log EC_{50} values were well correlated linearly with both the number of carbon atoms in the perfluoroalkyl chain and logKow predicted by EPI Suite v4.0. The authors therefore inferred that the toxicity of these chemicals is governed by the extent to which the biological membranes of algae are disrupted. However, the tested (nominal) concentrations of the solutions ranged from 0.000005 to 50 mM. This concentration range may induce micelle formation at higher concentrations. A summary of aquatic toxicity data to algae is given in Table 18.

Invertebrate Species

Water Flea

Some toxicity data of PFOS salts on *Daphnia magna* were summarized in an OECD document.[39] A lowest acceptable 48-hr EC_{50} value of 27 mg L^{-1} was reported for PFOS potassium salt, with two other 48-hr values of 58 and 61 mg L^{-1}. The 48-hr EC_{50} value of the lithium salt of PFOS was reported as 210 mg L^{-1}. Also, a 48-hr EC_{50} value of approximately 4.0 mg L^{-1} was determined for the didecyldimethyl–ammonium salt of PFOS (PFOSDDA) in water accommodated fractions of an aqueous mixture containing the substance. However, the actual exposure concentrations of PFOS were not determined in this test and it is possible that the didecyldimethylammonium may have contributed to the toxicity of the test medium. Sub-chronic/chronic toxicity was tested using survival, growth, and reproduction as endpoints over exposure periods of up to 28 days. NOECs of 12 and 7 mg L^{-1} were determined

TABLE 18. Aquatic toxicity of PFCs to algae with IC_{50} (mg L^{-1}) and NOEC (mg L^{-1})

Chemical	IC_{50}	NOEC	Endpoint	Organism	Reference
PFOSK	70	70	72-hr cell density	*P. subcapitata*	39
PFOSK	74	70	72-hr area under curve	*P. subcapitata*	39
PFOSK	120	70	72-hr growth rate	*P. subcapitata*	39
PFOSK	71	44	96-hr cell density	*P. subcapitata*	39
PFOSK	71	44	96-hr area under curve	*P. subcapitata*	39
PFOSK	126	44	96-hr growth rate	*P. subcapitata*	39
PFOSK	82		96-hr cell density	*P. subcapitata*	39
PFOSK	176	94	96-hr growth rate	*A. flos-aquae*	39
PFOSK	305	206	96-hr growth rate	*N. pelliculosa*	39
PFOSK	>3.2	>3.2	96-hr growth rate	*S. costatum*	39
PFOS	77.8		72-hr fluorescence	*S. obliquus*	116
PFOS	99.9		72-hr optical density	*S. obliquus*	116
PFOSK	48.2	5.3	96-hr cell density	*S. capricornutum*	110
PFOSK	59.2	16.6	96-hr Chlorophyll (*a*)	*S. capricornutum*	110
PFOSK	81.6	8.2	96-hr cell density	*C. vulgaris*	110
PFOSK	88.1	9.6	96-hr Chlorophyll (*a*)	*C. vulgaris*	110
PFHxA	4032		72-hr optical density	*C. vulgaris*	118
PFHpA	1896		72-hr optical density	*C. vulgaris*	118
PFOA	977.2		72-hr optical density	*C. vulgaris*	118
PFNA	496.6		72-hr optical density	*C. vulgaris*	118
PFHxA	1482		72-hr optical density	*S. marinoi*	118
PFHpA	873.7		72-hr optical density	*S. marinoi*	118
PFOA	368.5		72-hr optical density	*S. marinoi*	118
PFNA	194.9		72-hr optical density	*S. marinoi*	118
PFHxA	998.7		72-hr optical density	*G. amphibium*	118
PFHpA	517		72-hr optical density	*G. amphibium*	118
PFOA	248.4		72-hr optical density	*G. amphibium*	118
PFNA	129.9		72-hr optical density	*G. amphibium*	118
PFDoA	160.3		72-hr fluorescence	*S. obliquus*	116
PFDoA	112.4		72-hr cell density	*S. obliquus*	116
PFTeA	137.1		72-hr fluorescence	*S. obliquus*	116
PFTeA	95.7		72-hr cell density	*S. obliquus*	116
APFO		200	72-hr growth rate	*P. subcapitata*	115
APFO		12.5	96-hr growth rate	*P. subcapitata*	115

for *D. magna* reproduction in 21- and 28-day tests respectively. In the 21-day test the NOECs for survival and growth were also 12 mg L^{-1}, indicating that reproduction was no more sensitive than these two other endpoints.

Boudreau et al.[110] tested the toxicity of PFOSK on the invertebrates *Daphnia magna* and *Daphnia pulicaria* using standard laboratory protocols. *D. magna* showed more sensitive toward PFOSK than *D. pulicaria* with 48-hr immobility EC_{50} values of 67.2 and 134 mg L^{-1}, respectively. The 48-hr immobility NOEC values for *D. magna* and *D. pulicaria* were 0.8 and 13.6 mg L^{-1}, respectively. For adult survival, *D. magna* showed a 48-hr LC_{50} of 130 mg L^{-1}, whereas *D. pulicaria* had a 48-hr LC_{50} of 169 mg L^{-1}. Compared with the acute toxicity, the 21-day LC_{50} and lethality NOEC for *D. magna* were 42.9 and 5.3 mg L^{-1}, respectively.

Li[114] tested the toxicity of PFOSK and APFO to *Daphnia magna*. It was found that PFOSK was more toxic than APFO. Li reported that the 24- and 48-hr LC_{50}s for PFOSK exposures are 193 and 63 mg L^{-1}, respectively. These values are similar to the values of Boudreau et al.[110] The values for APFO exposure are larger than those of PFOS as 298 and 181 mg L^{-1}, respectively. Furthermore, Li[119] tested the chronic effects of APFO and PFOSK on three enzyme activities, survival and reproduction of *Daphnia Magna*. Based on the total number of neonates produced per female, the NOECs for reproduction of *D. magna* were 1 and 10 mg L^{-1} after 21 days of exposure to PFOA and PFOS, respectively. The 21-day LC_{50} and survival NOEC values of PFOA to *D. magna* were all greater than 100 mg L^{-1}, while the values were 9.1 and 5 mg L^{-1} for PFOS exposure. However, no significant changes in cholinesterase, catalase, and heme peroxidase activities were observed between controls and exposure concentrations, which may reveal that daphnids are not sensitive to APFO and PFOSK at the exposure concentrations.

Colombo et al.[115] evaluated acute and chronic freshwater aquatic toxicity of APFO on water flea *Daphnia magna* following the OECD test guidelines. The calculated 24- and 48-hr EC_{50} values were 599 and 480 mg L^{-1} APFO, respectively, based on nominal APFO concentrations and immobility. In the chronic reproduction test, no statistically significant mortality was observed in parent daphnids during the test, and the 21-day EC_{50} and NOEC values based on immobility of parent daphnids were greater than 88.6 mg L^{-1} APFO. Between the mean measured test concentrations of 9.16 and 88.6 mg L^{-1}, APFO was found to induce a delay in the appearance of the first brood, which occurred between days 8 and 16. APFO caused a decrease in the average number of broods per parent organism surviving to test termination with the mean number of broods decreasing from 4.75 to 1.22 in a concentration-dependent manner. A statistically significant inhibition of growth of parent daphnids at the end of the test was observed at 88.6 mg L^{-1} APFO. The 21-day EC_{50} for growth as length was calculated to be greater than 88.6 mg L^{-1} and the 21-day growth NOEC was 44.2 mg L^{-1} APFO.

Ji et al.[120] measured acute and chronic toxicity of PFOS and PFOA to two kinds of freshwater fleas, *Daphnia magna* and *Moina macrocopa*. In general, PFOS was more toxic than PFOA for these two organisms, and *M. macrocopa* exhibited greater sensitivity than *D. magna* to these two compounds in both acute and chronic exposures. The 24-hr EC_{50} for *D. magna* was 76.8 mg L^{-1} for PFOS and 675 mg L^{-1} for PFOA, while the 48-hr EC_{50}s were found to be 37.36 and 476.5 mg L^{-1}, respectively. For *M. macrocopa*, the 24-hr EC_{50} was 38.58 mg L^{-1} for PFOS and 348.8 mg L^{-1} for PFOA, while 48 hr EC_{50}s were 17.95 and 199.5 mg L^{-1}, respectively. The 21-day reproduction NOECs based on the number of offspring per adult *D. magna* determined for PFOS and PFOA were 1.25 and 12.5 mg L^{-1}, respectively. During a seven-day chronic toxicity test, *M. macrocopa* showed significant reproductive changes

at 0.31 mg L^{-1} for PFOS, which was approximately seven times lower than the effect concentrations observed over the 21-day exposure in *D. magna*.

Phillips et al.[113] performed 48-hr acute toxicity tests of FTCAs and FTUCAs on *Daphnia magna*. Endpoints were evaluated based on survival and immobility. Results revealed that *Daphnia magna* was particularly sensitive to the telomer acids of chain length 8 and 10 FCs, and more sensitive than *Chironomus tentan* and *Lemna gibba*. In addition, the saturated forms of the telomer acids were usually more toxic than their unsaturated counterparts, and the fluorotelomer acids were more toxic than the corresponding PFCAs. Phillips et al.[121] tested the chronic toxicity of 10:2 FTCA and 10:2 FTUCA to *Daphnia magna*. It was found that 10:2 FTCA was consistently more toxic than 10:2 FTUCA. LC_{50}s were 150 and >60 mg L^{-1} for 10:2 FTUCA and 10:2 FTCA, respectively. Reproduction was significantly reduced relative to the controls, with respective EC_{50}s for time to first brood and mean number of offspring/female of 287 and 214 mg L^{-1} for 10:2 FTUCA and 50 and 48 mg L^{-1} for 10:2 FTCA. Table 19 shows an overview of aquatic toxicity data of PFCs for water fleas.

Other Invertebrate Organisms

Results of toxicity studies of PFOSK on one species of freshwater mussel (*Unio complamatus*) and three species of saltwater invertebrate, *Mysidopsis bahia* (Mysid shrimp), *Crassostrea virginica* (Eastern oyster), and *Artemia* sp (Brine shrimp) were reported in an OECD document.[39] The 96-hr LC_{50} of PFOSK for the freshwater mussel, *U. complamatus* was determined to be 59 mg L^{-1} with the 96-hr NOEC of 20 mg L^{-1}. For the saltwater invertebrates, mortality during 96-hr exposure was assessed in tests with *Mysidopsis bahia* and *Artemia* sp, while reduction in shell deposition was measured over a 96-hr exposure period in a test with *C. virginica*. A 96-hr LC_{50} value of 3.6 mg L^{-1} and an associated NOEC of 1.1 mg L^{-1} were determined in the test with the Mysid shrimp *Mysidopsis bahia*. This is comparable to a 96-hr EC_{50} value of >3.0 mg L^{-1} for effects on shell deposition in the oyster and a 48-hr LC_{50} of 8.9 mg L^{-1} for mortality of the Brine shrimp. A 35-day chronic toxicity study was performed on the Mysid shrimp *Mysidopsis bahia*. The 35-day NOECs determined for survival, growth and reproduction in this test were 0.55, 0.25 and 0.25 mg L^{-1}, respectively.

MacDonald et al.[122] assessed the toxicity of PFOSK and PFOA to the aquatic midge *Chironomus tentans* under laboratory conditions. It was found that *C. tentans* is relatively insensitive to PFOA but highly sensitive to PFOSK. A preliminary test using PFOA indicated no significant impacts on survival or growth of *Chironomus tentans*. A 10-day assay with PFOSK concentrations ranging from 1 to 150 μg L^{-1} produced an EC_{50} of 87.2 $\pm$ 11.6 μg L^{-1} for growth. However, the LC_{50} for survival fell outside the range of test concentrations. Subsequently, the authors conducted a chronic life-cycle test using a nominal concentration range of 1 to 100 μg L^{-1} PFOSK. Three of

TABLE 19. Aquatic toxicity data of PFCs to water flea, showing as IC_{50}/LC_{50} and NOEC in units of mg L^{-1}

Chemical	48-hr LC_{50}	48-hr IC_{50}	48-hr NOEC	21-day EC_{50}	Organism	Reference
PFOSK		61	33 (I)		*D. magna*	39
PFOSK		27			*D. magna*	39
PFOSK		58			*D. magna*	39
PFOS Li^+		210	100 (I)		*D. magna*	39
PFOSDDA		4.0	2.2 (I)		*D. magna*	39
PFOSK				12 (repro)	*D. magna*	39
PFOSK				11 (28-day repro)	*D. magna*	39
PFOSK		63	20 (I)		*D. magna*	114
APFO		181	125 (I)		*D. magna*	114
APFO				>100	*D. magna*	119
PFOSK				9.1	*D. magna*	119
PFOA		476.5			*D. magna*	120
PFOS		37.36			*D. magna*	120
PFOA		199.5			*M. macrocopa*	120
PFOS		17.95			*M. macrocopa*	120
PFOSK	130	67.2	33.1 (L); 0.8 (I)	42.9	*D. magna*	110
PFOSK	169	134	46.9 (L); 13.6 (I)		*D. pulicaria*	110
APFO		480		>88.6	*D. magna*	115
8:2 FTUCA	8.45	4.01			*D. magna*	113
10:2 FTUCA	0.93	0.28			*D. magna*	113
8:2 FTCA	3.52	3.03			*D. magna*	113
10:2 FTCA	0.06	0.06			*D. magna*	113
10:2 FTUCA				0.150 (L)	*D. magna*	121
10:2 FTUCA				0.287 (TFB)	*D. magna*	121
10:2 FTUCA				0.214 (YN)	*D. magna*	121
10:2 FTCA				0.060 (L)	*D. magna*	121
10:2 FTCA				0.050 (TFB)	*D. magna*	121
10:2 FTCA				0.048 (YN)	*D. magna*	121

Note. L = lethality; TFB = time to first brood; YN = mean number of offspring produced per female reproduction day.

the four endpoints measured—survival, growth, and emergence—were significantly affected, with EC_{50} values of 92 ± 3, 94 ± 3, and 95 ± 3 μg L^{-1}, respectively. Reproduction was not affected at those PFOSK concentrations. PFOSK toxicity thresholds for *C. tentans* are as much as three orders of magnitude lower than those reported for other aquatic organisms, such as cladocerans *Daphnia magna* and *D. pulicaria*, aquatic macrophyte *Lemna gibba*, and the green algae *Pseudokirchneriella subcapitatum* and *Chlorella vulgaris*.

Phillips et al.[113] performed 10-day toxicity tests of FTCAs and FTUCAs on *Chironomus tentan*. Endpoints were evaluated based on survival and immobility at the end of the 10-day exposure. Growth was measured as ash-free dry weight (AFDW), which was determined as the mean difference between the dry weight (60°C for 24 hr) and ashed weight (550°C for 2 hr). Results revealed that *Chironomus tentan* was sensitive to the telomer acids

of chain length≥6 FCs, and toxicity increased with increasing chain length. In general, the saturated forms of the telomer acids were more toxic than their unsaturated counterparts, and the fluorotelomer acids were more toxic than the corresponding PFCAs. Phillips et al.[121] tested chronic toxicity of 8:2 FTCAs to *Chironomus dilutus*. EC_{50}s for survival and growth at 20 days were 2610 and 1250 mg L^{-1}, respectively. Total emergence and time to first emergence, the most sensitive endpoints, yielded EC_{50}s of 440 and 890 mg L^{-1}. Few adults emerged and no reproduction occurred at the two highest concentrations (600 and 1540 mg L^{-1}). The mean number of eggs/female was not affected.

Li[114] tested toxicities of PFOSK and APFO on four freshwater invertebrate species, water flea (*Daphnia magna*) planarian (*Dugesia japonica*), green neon shrimp (*Neocaridina denticulate*), and snail (*Physa acuta*). It was found that PFOSK was more toxic than APFO for all species tested. Values of the 48-hr LC_{50} of PFOSK for all test species ranged from 27 to 233 mg L^{-1} and values of the 96-hr LC_{50} for three of the species ranged from 10 to 178 mg L^{-1}. Values of the 48-hr LC_{50} of APFO for all test species ranged from 181 to 732 mg L^{-1} and values of the 96-hr LC_{50} for three of the species ranged from 337 to 672 mg L^{-1}. The most sensitive freshwater species to PFOSK was the green neon shrimp (*Neocaridina denticulate*) with a 96-hr LC_{50} of 10 mg L^{-1}, while the aquatic snail (*Physa acuta*) has the highest resistance to PFOSK or APFO toxicity among the aquatic organisms tested over each exposure period.

Wang et al.[123] tested the population growth impairment potential of FTOH on *Tetrahymena thermophila* with an open system (96-well microplates) and in a closed system (closed flasks). Furthermore, lactate dehydrogenase (LDH) leakage was measured to check the mode of action of direct membrane damage. The results revealed that no growth inhibition was found in either of the systems for 8:2 FTOH and 10:2 FTOH. However, 4:2 FTOH inhibited the population growth in the closed system with a 24-hr EC_{50} of 276.1 mg L^{-1}, whereas 6:2 FTOH had a 24-hr EC_{50} of 64.3 mg L^{-1} in the closed system. Macronucleus destruction was observed for 6:2 FTOH exposures. However, no direct membrane damage was detectable. Comparing the results from the two test systems, the authors suggested that tests in a closed system are more reliable for testing these volatile compounds with *Tetrahymena thermophila* than in an open system. Table 20 provides an overview of reported toxicity data of PFCs to invertebrate species, excluding the data for water flea.

Zooplankton Community

Sanderson et al.[124] determined the toxicological effects of PFOSK on freshwater zooplankton in 30-L indoor microcosms. PFOSK persisted in the water phase with only slight reductions of the concentration during the test; only a decrease of the actual concentration of 33.9 mg L^{-1} at day 1 to 29.8 mg

TABLE 20. Aquatic toxicity data of PFCs to other invertebrate organisms

Chemical	EC_{50}/LC_{50}	Endpoint	Organism	Reference
PFOSK	59 mg L^{-1}	96-hr Lethal	*Unio complamatus*	39
PFOSK	3.6 mg L^{-1}	96-hr Lethal	*Mysidopsis bahia*	39
PFOSK	>3.0 mg L^{-1}	96-hr reduction in shell deposition	*Crassostrea virginica*	39
PFOSK	8.9 mg L^{-1}	96-hr Lethal	*Artemia* sp	39
PFOSK	>150 μg L^{-1}	10-day Survival	*Chironomus tentans*	122
PFOSK	87.2 μg L^{-1}	10-day Growth	*Chironomus tentans*	122
PFOSK	92.2 μg L^{-1}	20-day Survival	*Chironomus tentans*	122
PFOSK	93.8 μg L^{-1}	20-day Growth	*Chironomus tentans*	122
PFOSK	94.5 μg L^{-1}	20-day emergence	*Chironomus tentans*	122
PFOSK	34 mg L^{-1}	24-hr Lethal	*Dugesia japonica*	114
PFOSK	27 mg L^{-1}	48-hr Lethal	*Dugesia japonica*	114
PFOSK	26 mg L^{-1}	72-hr Lethal	*Dugesia japonica*	114
PFOSK	23 mg L^{-1}	96-hr Lethal	*Dugesia japonica*	114
PFOSK	>200 mg L^{-1}	24-hr Lethal	*Neocaridina denticulate*	114
PFOSK	57 mg L^{-1}	48-hr Lethal	*Neocaridina denticulate*	114
PFOSK	20 mg L^{-1}	72-hr Lethal	*Neocaridina denticulate*	114
PFOSK	10 mg L^{-1}	96-hr Lethal	*Neocaridina denticulate*	114
PFOSK	271 mg L^{-1}	24-hr Lethal	*Physa acuta*	114
PFOSK	233 mg L^{-1}	48-hr Lethal	*Physa acuta*	114
PFOSK	208 mg L^{-1}	72 hr Lethal	*Physa acuta*	114
PFOSK	178 mg L^{-1}	96-hr Lethal	*Physa acuta*	114
APFO	352 mg L^{-1}	24-hr Lethal	*Dugesia japonica*	114
APFO	345 mg L^{-1}	48-hr Lethal	*Dugesia japonica*	114
APFO	343 mg L^{-1}	72-hr Lethal	*Dugesia japonica*	114
APFO	337 mg L^{-1}	96-hr Lethal	*Dugesia japonica*	114
APFO	>1000 mg L^{-1}	24-hr Lethal	*Neocaridina denticulate*	114
APFO	712 mg L^{-1}	48-hr Lethal	*Neocaridina denticulate*	114
APFO	546 mg L^{-1}	72-hr Lethal	*Neocaridina denticulate*	114
APFO	454 mg L^{-1}	96-hr Lethal	*Neocaridina denticulate*	114
APFO	856 mg L^{-1}	24-hr Lethal	*Physa acuta*	114
APFO	732 mg L^{-1}	48-hr Lethal	*Physa acuta*	114
APFO	697 mg L^{-1}	72-hr Lethal	*Physa acuta*	114
APFO	672 mg L^{-1}	96-hr Lethal	*Physa acuta*	114
6:2 FTCA	63.1 mg L^{-1}	10 days ash-free dry weight	*Chironomus tentan*	113
6:2 FTCA	75.2 mg L^{-1}	10 days Lethal	*Chironomus tentan*	113
8:2 FTCA	5.9 mg L^{-1}	10 days ash-free dry weight	*Chironomus tentan*	113
8:2 FTCA	12.4 mg L^{-1}	10 days Lethal	*Chironomus tentan*	113
10:2 FTCA	>16.3 mg L^{-1}	10 days ash-free dry weight	*Chironomus tentan*	113
10:2 FTCA	>16.3 mg L^{-1}	10 days Lethal	*Chironomus tentan*	113
8:2 FTUCA	16.6 mg L^{-1}	10 days ash-free dry weight	*Chironomus tentan*	113
8:2 FTUCA	21.2 mg L^{-1}	10 days Lethal	*Chironomus tentan*	113
10:2 FTUCA	6.43 mg L^{-1}	10 days ash-free dry weight	*Chironomus tentan*	113
10:2 FTUCA	8.43 mg L^{-1}	10 days Lethal	*Chironomus tentan*	113
8:2 FTCA	2.61 mg L^{-1}	20-day survival	*Chironomus dilutus*	121
8:2 FTCA	1.25 mg L^{-1}	20-day growth	*Chironomus dilutus*	121
8:2 FTCA	0.44 mg L^{-1}	Total% emergence	*Chironomus dilutus*	121
8:2 FTCA	0.89 mg L^{-1}	Time to first emergence	*Chironomus dilutus*	121
4:2 FTOH	276.1 mg L^{-1}	24-hr population growth inhibition	*Tetrahymena thermophila*	123
6:2 FTOH	64.3 mg L^{-1}	24-hr population growth inhibition	*Tetrahymena thermophila*	123

L^{-1} at day 35 was significant. PFOSK significantly reduced the zooplankton community at 10 mg L^{-1} after two to three weeks of treatment. However, no zooplankton $NOEC_{community}$ could be determined due to lack of statistical power at the 1 mg L^{-1} level. The authors concluded that the rank order of susceptibility for the test community was *Copepoda* > *Cladocera* > *Rotifera*, assuming all adverse direct effects.

Sanderson et al.[125] also tested the impact of PFOA on the structure of the zooplankton community using the same indoor microcosms. Some significant ($p < .01$) temporal fluctuations in zooplankton abundance were observed. These fluctuations, however, did not allow for derivation of a $NOEC_{community}$. Lowest observed effect concentration (LOEC) for various species varied between 10 and 70 mg L^{-1}. Based on the LOEC values derived, the following tentative order of descriptors sensitivity was obtained: *Daphnia magna* > richness > *Cyclops canthocamptus staphylinus* > *Cyclops diaptomus* > total zooplankton > *Rotifera* sp. The long-term ecological significance of these temporal fluctuations could not be determined, although the results showed that the structure of the ecosystem was changed from a more diverse community dominated by larger species toward a less diverse community dominated by smaller and more robust species.

After these tests, Sanderson et al.[126] compared the effects of the PFOA sodium salt and PFOSK on 30 L indoor microcosm to 12000 L outdoor microcosm experiments, with 225 mL single species laboratory tests as reference. It was found that PFOSK is more toxic to zooplankton than PFOA. With increasing concentrations the zooplankton community became simplified toward more robust rotifer species. Changes in species richness are statistically a more powerful endpoint than total abundance. The statistical power of the designs exhibited an inverse proportionality between complexity and realism, which was more powerful for the indoor microcosm as compared to the outdoor microcosm. It is surprising that the 30 L study had a lower LOEC value for *Daphnia magna* than the laboratory chronic test, indicating that laboratory tests are not always conservative relative to microcosm experiments. Table 21 provides an overview of aquatic toxicity data of PFOS and PFOA on zooplankton communities.

TABLE 21. Aquatic toxicity of PFOS and PFOA on zooplankton community (mg L^{-1})

Chemical	$LOEC_{community}$	Design	Reference
PFOSK	13-50 (*D. pulicaria*)	Laboratory	126
PFOSK	1-10	30 L indoor	126
PFOSK	10-30	12000 L outdoor	126
PFOA	—	Laboratory	126
PFOA	30-70	30 L indoor	126
PFOA	30-70	12000 L outdoor	126

TABLE 22. Aquatic toxicity of selected PFCs to *Vibrio fischeri* with 30 min. bioluminescence inhibition as the endpoint

Chemical	EC_{50}/LC_{50} (mg L^{-1})	Organism	Reference
PFHxA	1340	*Vibrio fischeri*	127
PFHpA	1099	*Vibrio fischeri*	127
PFOA	571.4	*Vibrio fischeri*	127
PFNA	532.8	*Vibrio fischeri*	127

Bacteria

Mulkiewicz et al.[127] tested the acute toxicity of PFHxA, PFHpA, PFOA, PFNA, and PFDA on the marine bacterium *Vibrio fischeri*. It was found that bioluminescence in *V. fischeri* bacteria was inhibited with respective EC_{50} values of 1340, 1099, 571.4, and 532.8 mg L^{-1} for PFHxA, PFHpA, PFOA, and PFNA, respectively. In addition, a hormetic response was also noticed in the *V. fischeri* test. There is a relationship between the toxicity of the PFCAs and the perfluorocarbon chain length: in the test system applied, the longer the perfluorocarbon chain, the more toxic the acids were found to be. Table 22 provides an overview of the data generated by these authors.

Amphibians

The OECD[39] reported an embryo teratogenesis assay carried out on PFOSK with *Xenopus laevis* (African clawed frog). Exposure of the embryos for 96 hr resulted in an LC_{50} of 13.8 mg L^{-1} for mortality and an EC_{50} of 12.1 mg L^{-1} for malformations. The minimum concentration that inhibited growth was 7.97 mg L^{-1}. The teratogenic index, calculated as the ratio of the 96-hr LC_{50} to the 96-hr EC_{50}, was found to be 1.1. The value indicates that PFOSK has a low potential to be a developmental hazard for this species.

Ankley et al.[101] tested the effects of PFOSK on survival and development of the northern leopard frog (*Rana pipiens*) from early embryogenesis through complete metamorphosis. Exposures were conducted via water at measured PFOSK concentrations ranging from 0.03 to 10 mg L^{-1}. Survival of *R. pipiens* was significantly decreased in the 10 mg L^{-1} treatment group within approximately two weeks of test initiation. Survival was not affected by PFOSK at lower concentrations; however, time to metamorphosis was delayed and growth reduced in the 3 mg L^{-1} treatment group. Mean LC_{50} values (95% confidence interval), based on measured PFOSK concentrations, were >12.5, 11.0 (8.35–14.2), 7.71 (6.14–9.07), 6.59 (5.52–7.74), and 6.21 (5.12–7.52) mg L^{-1} at week 1, 2, 3, 4, and 5 of the test, respectively. The developmental delay observed in tadpoles exposed to PFOSK could be related to alterations in thyroid function; however, further experiments are needed to assess this possibility. Table 23 provides an overview of aquatic toxicity data of PFOS and PFOA on amphibians.

TABLE 23. Aquatic toxicity of PFOS on amphibians

Chemical	LC_{50}/EC_{50} (mg L^{-1})	Endpoint	Organism	Reference
PFOSK	12.1/13.8	96-hr lethal/malformation	*Xenopus laevis*	39
PFOSK	>12.5	1-week lethal	*Rana pipiens*	101
PFOSK	11.0	2-week lethal	*Rana pipiens*	101
PFOSK	7.71	3-week lethal	*Rana pipiens*	101
PFOSK	6.59	4-week lethal	*Rana pipiens*	101
PFOSK	6.21	5-week lethal	*Rana pipiens*	101

Fish

The OECD[39] listed the toxicity data of PFOS and its salts on fishes submitted by 3M. Three species of freshwater fish, *Pimephales promelas* (Fathead minnow), *Lepomis macrochirus* (Bluegill sunfish), and *Oncorhynchus mykiss* (Rainbow trout), were tested for acute toxicity. *P. promelas* was the most susceptible freshwater fish species in acute tests with a lowest 96-hr LC_{50} of 4.7 mg L^{-1} for the lithium salt of PFOS. *L. macrochirus*, and *O. mykiss*, were only marginally less susceptible—a 96-hr LC_{50} value of 7.8 mg L^{-1} was determined for both species for the diethanolamine (DEA) and the potassium salt, respectively. Another test on *O. mykiss* showed an LC_{50} of 22 mg L^{-1} for the potassium salt of PFOS. A very high 96-hr LC_{50} value of approximately 200 mg L^{-1} was obtained for the didecyldimethylammonium salt of PFOS (PFOSDDA) following exposure of *P. promelas* in a test on water accommodated fractions of an aqueous mixture containing the substance. However, the actual exposure concentrations of PFOS were not determined and could not be estimated in this test. PFOS also exhibits acute toxicity to fish in saltwater. A 96-hr LC_{50} value of 13.7 mg L^{-1} was determined for the potassium salt in a test with *O. mykiss* acclimated to saltwater at a salinity of 30 parts per thousand. The data show saltwater acclimated *O. mykiss* to be of similar susceptibility to PFOS when compared with *O. mykiss* living in freshwater. However, in the absence of measured exposure concentrations, it should be noted that this study might have been conducted in excess of salt water solubility of the substance (2.5–20 mg L^{-1}, depending on salinity and purity). A further study using a saltwater fish, Sheepshead minnow (*Cyprinodon variegatus)* showed no toxicity up to 15 mg L^{-1} in the test.

Subchronic/chronic toxicity was tested on two species of freshwater fish—*Pimephales promelas* (Fathead minnow) and *Lepomis macrochirus* (Bluegill sunfish). Tests with *P. promelas* were designed to determine concentrations affecting early life stages of the fish over exposure periods of up to 42 days. Mortality data for *L. macrochirus* were obtained from a bioconcentration study in which deaths in the treated and control groups of fish were recorded over the 62-day uptake phase of the study. The lowest definitive NOEC of 0.3 mg L^{-1} was determined for *P. promelas* for the

potassium salt of PFOS. This value was found for both survival and growth endpoints. A test with *L. macrochirus* showed no significant mortality at an exposure concentration of 0.086 mg L^{-1} over a 62-day uptake phase, but 100% mortality was detected at a concentration of 0.87 mg L^{-1} after 35 days.

Ankley et al.[128] exposed sexually mature fathead minnow (*Pimephales promelas*) via the water for 21 days to 0 (control), 0.03, 0.1, 0.3, or 1 mg L^{-1} PFOSK and assessed effects on the reproductive capacity and endocrinology. To determine possible developmental effects, a subset of embryos from parental exposures at each test concentration, were kept for an additional 24 days at the same PFOSK treatment concentrations. A concentration of 1 mg L^{-1} PFOSK was lethal to adults within two weeks. The 21-day EC_{50} (95% confidence interval) for effects on fecundity of the fish was 0.23 (0.19~0.25) mg L^{-1}. Exposure to PFOSK caused various histopathological alterations, most prominently in ovaries of adult females. Adult males exposed to 0.3 mg L^{-1} PFOSK for 21 days exhibited decreased aromatase activity and elevated concentrations of plasma 11-ketotestosterone and testosterone. No significant adverse effects on survival or growth were observed in developing fathead minnows held for 24 days at PFOSK concentrations up to 0.3 mg L^{-1}.

Colombo et al.[115] tested the freshwater aquatic toxicity of APFO on embryo-larvae of rainbow trout, *Oncorhynchus mykiss*, following the OECD test guidelines. All trout exposed to a nominal APFO concentration of 1000 mg L^{-1} died within the first 24 hr of the test. The 96-hr LC_{50}, calculated by the binomial method, was 707 mg L^{-1} APFO based on nominal concentrations and mortality. The NOEC based on sub-ethal effects (changes in coloration, lethargy) at 96 hr was 125 mg L^{-1} APFO. APFO did not induce a significant increase in the mortality of trout embryos. The NOEC for mortality of embryos was 40 mg L^{-1} APFO based on the Dunnett's test. Measured APFO concentrations up to 40 mg L^{-1} did not delay the hatching period at any concentration relative to control. Nonparametric analyses of length and weight indicated that the NOEC for both endpoints was also 40 mg L^{-1} APFO.

Ji et al.[120] did two-generation fish toxicity tests of PFOA and PFOS on the teleost *Oryzias latipes*. The tests started with breeding and continued until 100 days after the hatching of offspring. The results showed that parental exposure to both compounds affected the performance of the offspring. Unexposed progeny-generation (F1) fish exhibited elevated mortality and histopathological changes that were correlated with exposure in the parental generation (F0). Continuous exposure from F0 through F1 generations increased the extent of adverse effects. The authors indicated that parental exposure to PFOS or PFOA may prevent or limit this transfer of the thyroid hormones to the fertilized eggs, thus limiting the available stock during embryogenesis and negatively affecting the development of embryos and the performance of larvae.

Huang et al.[129] studied the effect of PFOS on zebrafish embryos. Zebrafish embryos exhibited developmental toxicity of bent spine, uninflated swim bladder, decreased heart rate, and affected spontaneous movement after exposure to various PFOS concentrations (0~8 mg L^{-1}) from 6 to 120 hr postfertilization (hpf). The LC_{50} and the EC_{50} at 120 hpf were 2.20 and 1.12 mg L^{-1}, respectively. Continuous exposure to PFOS from 1 to 121 hpf resulted in a steady accumulation with no evidence of elimination. PFOS induced cell death at 24 hpf was consistently found in the brain, eye, and tail region of embryos. PFOS exposure induced lesions in the muscle fibers with histological examination. Behavior assessment of PFOS in zebrafish embryos elevated the basal rate of swimming after four days of exposure, and larvae exposed to PFOS (0.25~4 mg L^{-1}) for only 1 hr at 6 dpf swam faster with increasing PFOS concentration. Embryos/larvae exposed to 8 mg L^{-1} PFOS for 24-hr periods from 1 to 121 hpf showed the highest incidence of malformations in the 97~121 hpf window, being the most sensitive period for the development.

Hoff et al.[130] exposed common carp (*Cyprinus carpio*) to PFOS through a single intraperitoneal injection and assessed toxic effects in liver and serum after one and five days. After one day of exposure the average DNA basepair length (ABPL) was significantly increased in the 270 and 864 ng g^{-1} treatment groups. After five days of exposure significant increases relative to the control were observed for the 16270 and 864 ng g^{-1} treatment groups. At 561, 670, and 864 ng g^{-1} PFOS, a significant increase in serum alanine aminotransferase (ALT) activity became apparent after five days of exposure. This revealed enzyme leakage from the liver. For serum aspartate aminotransferase (AST) activity a significant increase for the 864 ng g^{-1} treatment group was observed with a value of 112% relative to the control. It was found that inflammation was not involved in the observed membranous enzyme leakage for the 561, 670, and 864 ng g^{-1} PFOS treatment groups. These results suggest that PFOS induces inflammation-independent enzyme leakage through liver cell membranes that might be related to cell necrosis. Furthermore, PFOS did not significantly affect serum antioxidant levels nor did it clearly induce peroxisome proliferation in carp.

Oakes et al.[131] investigated the effects of waterborne PFOS on oxidative stress and reproductive endpoints in five kinds of fishes, including fathead minnow (*Pimephales promelas*), rainbow trout (*Oncorhynchus mykiss*), creek chub (*Semotilus atromaculatus*), spottail shiner (*Notropis hudsonius*), and white sucker (*Catostomus commersoni*). In all fish species, 14~28 days exposure to PFOS produced only modest mortality at concentrations consistent with environmental spill scenarios. However, PFOS consistently increased hepatic fatty acyl–CoA oxidase activity and increased oxidative damage, as quantified using the 2-thiobarbituric acid–reactive substances assay. Vitellogenin was occasionally altered in the plasma with PFOS exposure, but responses varied with maturity. Oviposition frequency and egg deposition

in fathead minnow were not significantly impaired with PFOS exposure, despite a trend toward progressive impairment with increasing exposure concentrations.

Hagenaars et al.[132] exposed common carp (*Cyprinus carpio*) to PFOS through water for 14 days and evaluated its toxicity in the liver. Microarray data revealed that the expression of several genes in the liver was influenced by PFOS exposure and real-time PCR was used to confirm these gene expression changes. The affected genes were mainly involved in energy metabolism, reproduction, and stress response. Furthermore, the relative condition factor, the hepatosomatic index, and the available glycogen reserves of the exposed fish were significantly lower after 14 days of exposure than in the control fish. This suggests that there is a trade-off between the metabolic cost of toxicant exposure and processes vital to the survival of the organism.

Wei et al.[133] investigated the effects of waterborne PFOA on the expression of hepatic estrogen-responsive genes, vitellogenin (VTG), and estrogen receptor β (ERβ) and on the gonadal development in a freshwater rare minnow (*Gobiocypris rarus*). A significant increase of VTG expression in the livers of both mature males and females was observed after 14 and 28 days of exposure to 3, 10, and 30 mg L^{-1} PFOA, indicating that PFOA induces VTG synthesis. The expression of ERβ increased significantly in livers of both mature males and females after a 14-day exposure, although no difference was observed after a 28-day exposure. The development of oocytes in testes exposed to PFOA also provided evidence of estrogenic activity in males. The ovaries of PFOA-exposed females underwent degeneration, as reported in other fish species exposed to environmental estrogens. This preliminary study indicates that PFOA can disturb the activity of estrogen in mature male rare minnows by inducing hepatic estrogen-responsive genes, VTG and ERβ, and barrier female reproduction.

Liu et al.[134] identified two novel Cytochrome P450s (CYPs) in rare minnow *Gobiocypris rarus* and investigated the effects of waterborne PFOA on their corresponding mRNA levels in the gills of rare minnows. Upregulation of CYP3A mRNA was observed in the gills of male rare minnows exposed to 30 mg L^{-1} PFOA, while no significant changes occurred in exposed females. In contrast, downregulation of CYP1A mRNA was detected in the gills of male and female minnows exposed to PFOA. However, the effects of PFOA on gill mRNA levels of their potential regulators, aryl hydrocarbon receptor (AhR) for CYP1A, and pregnane X receptor (PXR) for CYP3A, were not consistent with the observed effects of PFOA on the corresponding CYP mRNA concentrations. This suggests a different or more complex transcriptional regulation of CYP expression following PFOA exposure. Liu et al.[135] isolated a full-length cDNA sequence (designated as CYP4T11) from rare minnow (*Gobiocypris rarus*) liver by rapid amplification of cDNA ends. It was found that CYP4T11 was predominantly expressed in liver and intestine with lower

expression in the gill and brain. PFOA exposure induced a consistent significant upregulation of both PPARα and PPARγ and a nonsignificant increase of CYP4T11 in the gill. In the liver, induced expression of PPARγ was observed, although no obvious changes in PPARα expression were observed. Induction of CYP4T11 was only observed in males at the highest concentration of PFOA. These results suggest that the PPAR-CYP4T11 signaling pathway may be involved in PFOA-induced gill toxicity, while the underlying mechanisms of action are more complex for the hepatotoxicity.

Liu et al.[136] investigated the effects of acute PFDoA exposure on the induction of oxidative stress and alteration of mitochondrial gene expression in the livers of female zebrafish (*Danio rerio*). They exposed female zebrafish to PFDoA via a single intraperitoneal injection and observed fishes after 48 hr, 96 hr, and 7 days of exposure. PFDoA-treated fish exhibited histopathological liver damage, including swollen hepatocytes, vacuolar degeneration, and nuclei pycnosis. Glutathione (GSH) content and catalase (CAT) activity decreased significantly at 48 hr postinjection while superoxide dismutase (SOD) activity was initially decreased at 48 hr postinjection but was then elevated by seven days postinjection. The activity of glutathione peroxidase (GPx) increased after 48 hr and after seven days compared to control fish, although the increased level at seven days postinjection was decreased compared to the level at 48 hr postinjection. Lipid peroxidation levels were increased at seven days postinjection, while no apparent induction was observed at 48 or 96 hr postinjection. The mRNA expression of medium-chain fatty acid dehydrogenase (MCAD) was induced, while the transcriptional expression of liver fatty acid binding protein (L-FABP), peroxisome proliferating activating receptor α (PPARα), carnitine palmitoyl-transferase I (CPT-I), uncoupling protein 2 (UCP-2), and Bcl-2 were significantly inhibited. Furthermore, the transcriptional expression of peroxisomal fatty acyl-CoA oxidase (ACOX), very long-chain acyl-CoA dehydrogenase (VLCAD), long-chain acyl-CoA dehydrogenase (LCAD) did not exhibit significant changes following PFDoA treatment. No significant changes were noted in the transcriptional expression of genes involved in mitochondrial respiratory chain and ATP synthesis, including the cytochrome *c* oxidase subunit I (COXI), the NADH dehydrogenase subunit I (NDI), and the ATP synthase F0 subunit 6 (ATPo6). These results demonstrated that PFDoA induced the turbulence of fatty acid β-oxidation and oxidative stress responses in the liver of female zebrafish.

Wei et al.[137] employed two-dimensional electrophoresis coupled with mass spectrometry to identify proteins differentially expressed in the livers of rare minnow (*Gobiocypris rarus*) following PFOA exposure at 3, 10, and 30 mg L^{-1}. After comparison of the protein profiles from treated and control groups, 34 and 48 protein spots were found altered in abundance (>twofold) from males and females, respectively. Matrix-assisted laser desorption/ionization (MALDI) tandem time-of-flight mass spectrometry (TOF/TOF) analysis identified 25 spots, corresponding to 22 different

proteins. These proteins were involved in intracellular fatty acid transport, oxidative stress, macromolecule catabolism, the cell cycle, maintenance of intracellular Ca^{2+} homeostasis, and mitochondrial function. In addition, they described gender differences in response to PFOA exposure from the comparison of the male and female protein profiles. Transcriptional analysis of nine mRNAs encoding proteins altered by PFOA in the proteome analysis was determined by real-time PCR. The consistent and discrepant results between mRNA and protein levels suggested that complicated regulatory mechanisms of gene expression were implicated in the response to PFOA exposure.

Wei et al.[138] investigated the hepatic gene expression profile in male and female rare minnows (*Gobiocypris rarus*) after exposure to 10 mg L^{-1} PFOA during 28 days using a custom cDNA microarray containing 1,773 unique genes. A total of 124 and 171 genes were significantly altered by PFOA in males and females, respectively, of which 43 genes were commonly regulated in both sexes. The affected genes are involved in multiple biological processes, including lipid metabolism and transport, hormone action, immune responses, and mitochondrial functions. PFOA exposure significantly suppressed genes involved in fatty acid biosynthesis and transport but induced genes associated with intracellular trafficking of cholesterol. Alterations in expression of genes associated with mitochondrial fatty acid β-oxidation were only observed in female rare minnows. In addition, PFOA inhibited genes responsible for thyroid hormone biosynthesis and significantly induced estrogen-responsive genes. These findings indicted that PFOA has the effect of endocrine disruption.

In a subsequent contribution, Wei et al.[139] detected changes of gene expression profiles in primary cultured hepatocytes of rare minnows exposed to six individual PFCs (PFOA, PFNA, PFDA, PFDoA, PFOS, and 8:2 FTOH) and four formulations of the PFCs mixtures. It was found that mixtures as well as individual compounds consistently regulated a particular gene set, which suggests that these conserved genes may play a central role in the toxicity mediated by PFCs. A number of genes regulated by the mixtures were identified in this study, while these genes were not affected by exposure to any single component. These genes are implicated in multiple biological functions and processes including metabolism and transport of fatty acid, metabolism of xenobiotics, immune responses, and oxidative stress. More than 80% of the altered genes in the PFOA- and PFOS-dominant mixture groups were of the same gene set, while the gene expression profiles from single PFOA and PFOS exposures were not as similar.

Liu et al.[140] studied the cellular toxicology of PFOA and PFOS in primary cultured hepatocytes of freshwater tilapia (*Oreochromis niloticus*) for 24 hr. Significant induction of reactive oxygen species (ROS) accompanied by increases in activities of superoxide dismutase (SOD), catalase (CAT), and glutathione reductase (GR) were found, while activities of glutathione

peroxidase (GPx) and glutathione-*S*-transferase (GST) were decreased. Glutathione (GSH) content was reduced following treatment of PFOA and PFOS. A dose-dependent increase in the lipid peroxidation (LPO) level (measured as maleic dialdehyde [MDA]) was observed only in the PFOA exposure groups, whereas LPO remained unchanged in the PFOS exposure groups. Furthermore, a significant activation of caspase-3, -8, and -9 activities was evident in both PFOS and PFOA exposure groups. Typical DNA fragmentation (DNA laddering) was further characterized by agarose gel electrophoresis. The overall results demonstrated that PFOS and PFOA are able to produce oxidative stress and induce apoptosis with involvement of caspases in primary cultured tilapia hepatocytes.

Liu et al.[141] investigated estrogenic activities of PFOA, PFOS, 4:2 FTOH, 6:2 FTOH, and 8:2 FTOH using vitellogenin (VTG) induction in primary cultured hepatocytes of freshwater male tilapia (*Oreochromis niloticus*). A dose-dependent induction of VTG was observed in 17-estradiol (E2), 4-nonylphenol (4-NP), and PFOSK-, PFOA-, and 6:2 FTOH-treated cells, whereas VTG levels remained unchanged in the 4:2 FTOH and 8:2 FTOH exposure groups at the concentrations tested. The estimated 48-hr EC_{50} values for E2, 4-NP, PFOSK, PFOA, and 6:2 FTOH were 0.128, 1.56, 8.07, 12.01, and 10.19 mg L^{-1}, respectively. Coexposure to binary mixtures of individual PFCs and E2 for 48 hr significantly inhibited E2-induced hepatocellular VTG production in a dose-dependent manner except for 4:2 FTOH. This indicated that PFCs showed anti-estrogenic effects together with E2. When coexposed with tamoxifen (an estrogen receptor inhibitor) for 48 hr, tamoxifen significantly inhibited the ability of these chemicals to stimulate vitellogenesis. This suggests that the estrogenic effect of PFCs is mediated by the estrogen receptor pathway in primary cultured tilapia hepatocytes.

Based on these results, Liu et al.[142] investigated the mechanism of estrogenic activity of 6:2 FTOH on 18-week-old zebrafish (*Danio rerio*) following a seven-day exposure. Exposure to 6:2 FTOH significantly increased plasma estradiol (E2) and testosterone (T) levels in both males and females. Furthermore, the ratio of T/E2 was reduced in females while it increased in males. In females, the increase of E2 was accompanied by the upregulated hepatic estrogenic receptor α (ERα) and vitellogenin (VTG1 and VTG3) expression. In males, the elevation of the T level is consistent with the upregulation of cytochrome P450 c17α-hydroxylase, 17,20-lyase (CYP17), and the downregulation of cytochrome P450 aromatase A (CYP19A). The authors concluded that exposure alters plasma sex hormone levels and gene transcription in the hypothalamic–pituitary–gonadal (HPG) axis of zebrafish. After that, Liu et al.[143] studied endocrine disruption and reproductive impairment of 8:2 FTOH on 4-month-old zebrafish. The plasma testosterone (T) and estradiol (E2) levels were significantly increased in the females, while T and E2 levels were decreased and increased in the males, respectively. The average number of eggs spawned and sperm production were reduced upon exposure

to the chemical, which also resulted in thinning of the eggshell and reduced protein content and egg diameter. Histological examination showed promotion of oocyte maturation and retarded spermination in female and male fish, respectively. Some gene transcriptions related with sex hormones were significantly regulated. In addition, exposure of adult zebrafish to 8:2 FTOH caused reduced hatching rates in the offspring. It was therefore concluded that exposure to 8:2 FTOH caused disruption of sex hormone biosynthesis and impaired reproduction in adult zebrafish, ultimately resulting in decreased hatching rates in the offspring.

Shi et al.[144] tested developmental toxicity and alteration of gene expression in zebrafish embryos exposed to different PFOS concentrations. Hatching was delayed and hatching rates as well as larval survivorship were significantly reduced after embryos were exposed to 1, 3, and 5 mg L^{-1} PFOS until 132 hpf. Developmental malformations were displayed, including epiboly deformities, hypopigmentation, yolk sac edema, tail and heart malformations, and spinal curvature upon exposure to PFOS concentrations of 1 mg L^{-1} or greater. Growth (body length) was significantly reduced in the 3 and 5 mg L^{-1} PFOS-treated groups. It was found that more apoptotic cells were present in the PFOS-treated embryos than in the control embryos and certain genes related to cell apoptosis were significantly regulated. In addition, some marker genes related to early thyroid development and the balance of androgens and estrogens were also significantly altered.

Shi et al.[145] analyzed the differential expression of proteins after zebrafish embryos were exposed to 0.5 mg L^{-1} PFOS 192 hr post fertilization using two-dimensional gel electrophoresis (2-DE) and peptide mass fingerprinting (PMF). Compared to the control, 69 proteins showed altered expression in the treatment group. Of the 69 spots corresponding to the proteins with altered expression, 38 were selected and subjected to matrix-assisted laser desorption/ionization tandem time-of-flight mass spectrometry (TOF/TOF) analysis; 18 proteins were identified in that analysis. These proteins were categorized into diverse functional classes such as detoxification, energy metabolism, lipid transport/steroid metabolic process, cell structure, signal transduction, and apoptosis. Shi et al.[146] subsequently investigated the expression of functionally relevant genes associated with the pathways of the hypothalamus–pituitary–thyroid (HPT) axis and the levels of thyroid hormones in zebrafish larvae under exposure to various PFOS concentrations. It was found that the expression of several genes in the HPT system was significantly regulated and whole body triiodothyronine (T_3) levels significantly increased although the thyroxine (T_4) content remained unchanged. It was inferred that PFOS exposure could alter gene expression in the HPT axis and that mechanisms of disruption of thyroid status by PFOS could occur at several steps in the synthesis, regulation, and action of thyroid hormones.

Du et al.[147] investigated chronic effects of water-borne PFOS exposure on growth, survival, and hepatotoxicity in zebrafish. Zebrafish fry (F_0, 14 days

postfertilization, dpf) were exposed 70 day to 0 (control), 10, 50, and 250 μg L^{-1} PFOS, followed by a further 30-day recovery in clean water. Although growth suppression (weight and length) was observed in fish treated with high concentrations PFOS during the exposure period, no mortality was observed throughout the 70-day experiment. Embryos and larvae (F1) derived from maternal exposure suffered malformation and mortality. Exposure to 50 and 250 μg/L PFOS could inhibit the growth of the gonads (GSI) in the female zebrafish. Histopathological alterations, primary with lipid droplets accumulation, were most prominently seen in the liver of males and the changes were not reversible, even after the fish were allowed to recover for 30 days in clean water. Hepatic vitellogenin (VTG) gene expression was significantly upregulated in both male and female zebrafish, but the sex ratio was not altered. The authors concluded that lower concentrations of PFOS in maternal exposure could result in offspring deformation and mortality.

Fang et al.[148] investigated the impact of PFOA on the expression of apolipoprotein genes in rare minnow (*Gobiocypris rarus*) after 14 days of exposure. Results showed that six apolipoprotein genes and their possible upstream genes, PPARα, PPARγ, and HNF4α, were significantly altered for some PFOA treated groups. It was inferred that these changes in gene expression may further influence lipid metabolism or other physiological functions in rare minnow. Aquatic toxicity data of PFCs to fish are summarized in Table 24.

SUMMARY

It is clear that PFCs are ubiquitous in the world and have the potential of causing toxicity to biota and plants. However, the data for their basic physicochemical properties are still scarce, and there are big debates regarding the actual values for some of the properties investigated. Discrepancies observed among studies may be related with problems of purity of chemicals, suited analytical methods, solubilities and aggregation of chemicals, dissociation in water, and sorption to the wall of containers. All of these issues may affect the measurement of the basic properties of PFCs. Only when these problems have been studied and solved, correct and recognized data can be obtained. Based on these data, we can get a clearer insight into the behavior and fate of PFCs in the environment.

It is nevertheless clear that due to their unique physicochemical characteristics, some PFCs tend to persist in surface water. So their potential adverse effects on aquatic organisms should be considered with priority. Some tests have been conducted on algae, aquatic plants, invertebrates, bacteria, amphibians, and fish. Although results showed that currently known PFC levels in surface water have no acute harmful impact on aquatic organisms, some PFCs (including the mostly studied PFC:PFOSK) have long-term adverse effects on aquatic organisms. Therefore, more attention and further research

TABLE 24. Overview of aquatic toxicity data of PFCs to fish (mg L^{-1})

Chemical	LC_{50}/EC_{50} (mg L^{-1})	NOEC (mg L^{-1})	Medium	Fish	Reference
PFOSK	9.5(96*hr*)/–	3.3 (96 hr)	Freshwater	*Pimephales promelas*	39
PFOS Li^+	4.7(96*hr*)/–		Freshwater	*Pimephales promelas*	39
PFOSDDA	200(96*hr*)/–	> 170 (96 hr)	Freshwater	*Pimephales promelas*	39
PFOS DEA salt	7.8(96*hr*)/–	4.5 (96 hr)	Freshwater	*Lepomis macrochirus*	39
PFOSK	7.8(96*hr*)/–		Freshwater	*Oncorhynchus mykiss*	39
PFOSK	22(96*hr*)/–		Freshwater	*Oncorhynchus mykiss*	39
PFOSK	> 15(96*hr*)/–		Saltwater	*Cyprinodon variegatus*	39
PFOSK	13.7(96*hr*)/–		Saltwater	*Oncorhynchus mykiss*	39
PFOSK		0.3 (42-day survival and growth)	Freshwater	*Pimephales promelas*	39
PFOSK		>0.086, > 0.87 (62-day mortality)	Freshwater	*Lepomis macrochirus*	39
PFOSK	–/0.23(21 – *dayreproduce*)		Freshwater	*Pimephales promelas*	128
APFO	707(96*hr*)/–	125 (96 hr)	Freshwater	*Oncorhynchus mykiss*	115
PFOS	2.20/1.12(120*hpf*)		Freshwater	zebrafish embryo	129

are needed to assess the toxicity of PFCs toward aquatic organisms, not only for freshwater organisms but also for saltwater organisms. Research priorities for the future include (a) the toxicity assessment of emerging PFCs on aquatic organisms, such as perfluorinated phosphonic acids, FTOH acid, and polyfluorinated iodides; (b) toxicity assessment of branched isomer of PFCs; (c) study of mixture toxicity of PFCs and their derivates; (d) study of the toxicity of mixtures of PFCs and other non-PFC pollutants; and (e) assessment of the possible mode of action of PFCs in aquatic organisms and possible differences in mode of action between species. Following completion of these research efforts, we will most certainly have a clear and unbiased insight into the fate and effects of PFCs.

NOMENCLATURE

4:2 FTOH	1H,1H,2H,2H-perfluoro-1-hexanol
6:2 FTOH	1H,1H,2H,2H-perfluoro-1-octanol
8:2 FTOH	1H,1H,2H,2H-perfluoro-1-decanol
10:2 FTOH	1H,1H,2H,2H-perfluoro-1-dodecanol
BSAFs	Biota sediment accumulation factors
CAT	Catalase
CMC	Critical micelle concerntration
ECF	Electrochemical fluorination
FAs	Fatty acids
FTCAs	Fluorotelomer carboxylic acids
FTOHs	Fluorotelomer alcohols
FTUCAs	Unsaturated fluorotelomer carboxylic acids
GPx	Glutathione peroxidase
GR	Glutathione reductase
GSH	Glutathione
GST	Glutathione-*S*-transferase
HLC	Henry's law constant
LC_{50}	Median lethal concentration, 50%
L-PFOS	Linear PFOS
MDA	Maleic dialdehyde
MM-PFOS	Monomethyl-substituted PFOS
N-EtFOSA	N-ethyl perfluorooctane sulfonamide
N-EtFOSAA	N-ethyl perfluorooctane sulfonamido acetic acid
N-EtFOSE	N-ethyl perfluorooctane-sulfonamidoethanol
N-MeFOSA	N-methyl perfluorooctane-sulfonamide
N-MeFOSE	N-methyl perfluorooctane-sulfonamidoethanol
N-MeFOSEA	N-methyl perfluorooctane sulfonamidethylacrylate
NOEC	No observed effect concentration
PBDEs	Polybrominated diphenyl ethers

PCBs	Polychlorinated biphenyls
PCDD/Fs	Polychlorinated dibenzo-p-dioxin and dibenzofurans
PFAAs	Perfluoroalkyl acids
PFBA	Perfluorobutanoic acid
PFBS	Perfluorobutane sulfonate
PFBSK	Potassium perfluorobutane sulfonate
PFCs	Poly- and perfluorinated compounds
PFCAs	Perfluorinated carboxylic acids
PFDA	Perfluorodecanoic acid
PFDS	Perfluorodecane sulfonate
PFDoA	Perfluorododecanoic acid
PFDPA	Perfluorodecylphosphonic acid
PFHpA	Perfluoroheptanoic acid
PFHpS	Perfluoroheptane sulfonate
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonate
PFNA	Perfluorononanoic acid
PFNS	Perfluorononane sulfonate
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate
PFOSA	Perfluorooctane sulfonamide
PFOSF	Perfluorooctane sulfonyl fluoride
PFOSK	Potassium perfluorooctane sulfonate
PFPeA	Perfluoropentanoic acid
PFPeS	Perfluoropentane sulfonate
PFPrA	Perfluoropropionic acid
PFSAs	Perfluorinated sulfonate acids
PFTeDA	Perfluorotetradecanoic acid
PFTriDA	Perfluorotridecanoic acid
PFUnDA	Perfluoroundecanoic acid
POP	Persistent organic pollutant
PPARα	Peroxisome proliferating activating receptor α
ROS	Reactive oxygen species
SOD	Superoxide dismutase
TFA	Trifluoroacetic acid
VTG	Vitellogenin

ACKNOWLEDGMENTS

Guanghui Ding was supported by a grant of NUFFIC (C&B-CHN.09/11). The review was performed within the framework of the European Union supported FP7-project CADASTER: FP7-ENV-2007-1-212668.

REFERENCES

[1] Renner, R. (2001). Growing concern over perfluorinated chemicals. *Environmental Science and Technology*, 35, 154A–160A.
[2] Renner, R. (2003). Concerns over common perfluorinated surfactant. *Environmental Science and Technology*, 37, 201A–202A.
[3] Rayne, S., and Forest, K. (2009). Perfluoroalkyl sulfonic and carboxylic acids: A critical review of physicochemical properties, levels and patterns in waters and wastewaters, and treatment methods. *Journal of Environmental Science and Health, Part A*, 44, 1145–1199.
[4] Key, B. D., Howell, R. D., and Criddle, C. S. (1997). Fluorinated organic in the biosphere. *Environmental Science and Technology*, 31, 2445–2454.
[5] Kissa, E. (2001). *Fluorinated surfactants and repellants* (2nd ed.). New York: Marcel Decker.
[6] Giesy, J. P., and Kannan, K. (2002). Perfluorochemical surfactants in the environment. *Environmental Science and Technology*, 36, 146A–152A.
[7] Lau, C., Butenhoff, J. L., and Rogers, J. M. (2004). The developmental toxicity of perfluororalkyl acids and their derivatives. *Toxicology and Applied Pharmacology*, 198, 231–241.
[8] Schultz, M., Barofsky, D., and Field, J. (2003). Fluorinated alkyl surfactants. *Environmental Engineering Science*, 20, 487–501.
[9] Lehmler, H. (2005). Synthesis of environmentally relevant fluorinated surfactants: A review. *Chemosphere* 58, 1471–1496.
[10] Prevedouros, K., Cousins, I. T., Buck, R. C., and Korzeniowski, S. H. (2006). Sources, fate and transport of perfluorocarboxylates. *Environmental Science and Technology*, 40, 32–44.
[11] Paul, A. G., Jones, K. C., and Sweetman, A. J. (2009). A first global production, emission, and environmental inventory for perfluorooctane sulfonate. *Environmental Science and Technology*, 43, 386–392.
[12] 3M. (1999). *Fluorochemical use, distribution and release overview*. AR226-0550. St. Paul, MN: 3M Company.
[13] Powley, C. R., George, S. W., Russell, M. H., Hoke, R. A., and Buck, R. C. (2008). Polyfluorinated chemicals in a spatially and temporally integrated food web in the Western Arctic. *Chemosphere* 70, 664–672.
[14] Ellis, D. A., Martin, J. W., De Silva, A. O., Mabury, S. A., Hurley, M. D., Sulbaek Andersen, M. P., and Wallington, T. J. (2004). Degradation of fluorotelomer alcohols: A likely atmospheric source of perfluorinated carboxylic acids. *Environmental Science and Technology*, 38, 3316–3321.
[15] Martin, J. W., Mabury, S. A., and O'Brien, P. J. (2005). Metabolic products and pathways of fluorotelomer alcohols in isolated rat hepatocytes. *Chemico-Biological Interactions*, 155, 165–180.
[16] Wang, N., Szostek, B., Buck, R. C., Folsom, P. W., Sulecki, L., and Gannon, J. T. (2009). 8–2 Fluorotelomer alcohol aerobic soil biodegradation: pathways, metabolites, and metabolite yields. *Chemosphere*, 75, 1089–1096.
[17] Liu, J. X., Wang, N., Szostek, B., Buck, R. C., Panciroli, P. K., Folsom, P. W., Sulecki, L. M., and Bellin, C. A. (2010). 6–2 Fluorotelomer alcohol aerobic biodegradation in soil and mixed bacterial culture. *Chemosphere*, 78, 437–444.

[18] 3M. (2000). *Sulfonated perfluorochemicals in the environment: Sources, dispersion, fate and effects* (AR226-0545). St. Paul, MN: 3M Company.
[19] 3M. (2000). *Sulfonated perfluorochemicals: U.S. release estimation: 1997 Part 1: Life-cycle waste stream estimates.* AR226-0681. St. Paul, MN: 3M Company.
[20] Lau, C., Lindstrom, A. B., and Seed, J. (2009). Perfluorinated chemicals 2008: PFAA days II meeting report and highlights. *Reproductive Toxicology*, 27, 429–434.
[21] Giesy, J. P., and Kannan, K. (2001). Global distribution of perfluorooctane sulfonate in wildlife. *Environmental Science and Technology*, 35, 1339–1342.
[22] Houde, M., Martin, J. W., Letcher, R. J., Solomon, K. R., and Muir, D. C. G. (2006). Biological monitoring of polyfluoroalkyl substances: A review. *Environmental Science and Technology*, 40, 3463–3473.
[23] Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., and Seed, J. (2007). Perfluoroalkyl acids: A review of monitoring and toxicological findings. *Toxicological Sciences*, 99, 366–394.
[24] Dreyer, A., Weinberg, I., Temme, C., and Ebinghaus, R. (2009). Polyfluorinated compounds in the atmosphere of the Atlantic and Southern Oceans: Evidence for a global distribution. *Environmental Science and Technology*, 43, 6507–6514.
[25] Kannan, K., Corsolini, S., Falandysz, J., Fillmann, G., Kumar, K. S., Loganathan, B. G., Mohd, M. A., Olivero, J., Van Wouwe, N., Yang, J. H., and Aldous, K. M. (2004). Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. *Environmental Science and Technology*, 38, 4489–4495.
[26] D'eon, J. C., Crozier, P. W., Furdui, V. I., Reiner, E. J., Libelo, E. L., and Mabury, S. A. (2009). Observation of a commercial fluorinated material, the polyfluoroalkyl phosphoric acid diesters, in human sera, wastewater treatment plant sludge, and paper fibers. *Environmental Science and Technology*, 43, 4589–4594.
[27] Fromme, H., Tittlemier, S. A., Völkel, W., Wilhelm, M., and Twardella, D. (2009). Perfluorinated compounds–exposure assessment for the general population in western countries. *International Journal of Hygiene and Environmental Health*, 212, 239–270.
[28] Jones, P. D., Hu, W., De Coen, W., Newsted, J. L., and Giesy, J. P. (2003). Binding of perfluorinated fatty acids to serum proteins. *Environmental Toxicology and Chemistry*, 22, 2639–2649.
[29] Luebker, D. J., Hansen, K. J., Bass, N. M., Butenhoff, J. L., and Seacat, A. M. (2002). Interactions of fluorochemicals with rat liver fatty acid-binding protein. *Toxicology*, 176, 175–185.
[30] Martin, J. W., Mabury, S. A., Solomon, K. R., and Muir, D. C. (2003). Dietary accumulation of perfluorinated acids in juvenile rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry*, 22, 189–195.
[31] Martin, J. W., Mabury, S. A., Solomon, K. R., and Muir, D. C. G. (2003). Bioconcentration and tissue distribution of perfluorinated acids in rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry*, 22, 196–204.
[32] Kennedy, G. L., Butenhoff, J. L., Olsen, G. W., O'Connor, J. C., Seacat, A. M., Perkins, R. G., Biegel, L. B., Murphy, S. R., and Farrar, D. G. (2004).

The toxicology of perfluorooctanoate. *Critical Reviews in Toxicology*, 34, 351–384.

[33] Andersen, M. E., Butenhoff, J. L., Shang, S. C., Farrar, D. G., Kennedy, G. L., and Lau, C. (2008). Perfluoroalkyl acids and related chemistries—toxicokinetics and modes of action. *Toxicological Sciences*, 102, 3–14.

[34] Jensen, A. A., and Leffers, H. (2008). Emerging endocrine disrupters: Perfluoroalkylated substances. *International Journal of Andrology*, 31, 161–169.

[35] Olsen, G. W., Butenhoff, J. L., and Zobel, L. R. (2009). Perfluoroalkyl chemicals and human fetal development: An epidemiologic review with clinical and toxicological perspectives. *Reproductive Toxicology*, 27, 212–230.

[36] Shinoda, K., Hato, M., and Hayashi, T. (1972). The physico-chemical properties of aqueous solutions of fluorinated surfactants. *The Journal of Physical Chemistry*, 76, 909–914.

[37] Kunieda, H., and Shinoda, K. (1976). Krafft points, critical micelle concentrations, surface tension, and solubilizing power of aqueous solutions of fluorinated surfactants. *The Journal of Physical Chemistry*, 80, 2468–2470.

[38] NICNAS. (2005). *Potassium perfluorobutane sulfonate: Hazard assessment. Australia.* Retrieved from http://www.nicnas.gov.au/Publications/CAR/Other/Potassium_Perfluorobutane_Sulfonate_PDF.pdf

[39] Organization for Economic Cooperation and Development. (2002). *Hazard assessment of perfluorooctane sulfonate (PFOS) and its salts.* ENV/JM/RD(2002)17/FINAL. Joint Meeting of the Chemicals Committee and the Working Party on Chemicals, Pesticides, and Biotechnology, Environment Directorate. Paris: OECD. Retrieved from http://www.oecd.org/dataoecd/23/18/2382880.pdf

[40] Minnesota Pollution Control Agency. (2007). *Surface water quality criterion for perfluorooctane sulfonic acid.* STS Project 200604796.

[41] 3M. (2001). *Solubility of PFOS in water.* 3M Environmental Laboratory, Project Number E00-1716. St. Paul, MN: 3M Company.

[42] 3M. (2001). *Solubility of PFOS in natural seawater and an aqueous solution of 3.5% sodium chloride.* 3M Environmental Laboratory, Lab Project Number E00-1716. St. Paul, MN: 3M Company

[43] 3M. (2001). *Solubility of PFOS in octanol.* 3M Environmental Laboratory, Laboratory Project Number E00-1716. St. Paul, MN: 3M Company

[44] 3M Environmental Laboratory. (2001). *Characterization study of PFOA (lot #332), primary standard: Test control reference #TCR-99030–030. Phase: Solubility Determination.* St. Paul, MN: 3M Company

[45] Kaiser, M. A., Cobranchi, D. P., Kao, C. P. C., Krusic, P. J., Marchione, A. A., and Buck, R. C. (2004). Physicochemical properties of 8–2 fluorinated Telomer B alcohol. *Journal of Chemical and Engineering Data*, 49, 912–916.

[46] Liu, J., and Lee, L. S. (2005). Solubility and sorption by soils of 8:2 fluorotelomer alcohol in water and cosolvent systems. *Environmental Science and Technology*, 39, 7535–7540.

[47] Liu, J., and Lee, L. S. (2007). Effect of fluorotelomer alcohol chain length on aqueous solubility and sorption by soils. *Environmental Science and Technology*, 41, 5357–5362.

[48] Fischer-Drowos, S. G., Betz, L. R., Dibussolo, J. M., Miscavige, J. A., and Madhoun, N. M. (2007). Melting point and aqueous solubility determination of 2H-hexadecafluoro-2-decenoic acid ($C_{10}F_{16}H_2O_2$). *Journal of Chemical and Engineering Data*, 52, 759–761.

[49] Bhhatarai, B., and Gramatica, P. (2010). Prediction of aqueous solubility, vapor pressure and critical micelle concentration for aquatic partitioning of perfluorinated chemicals. *Environmental Science and Technology*, 45, 8120–8128.

[50] Crowder, G. A., Taylor, Z. L., Reed, T. M., and Young, J. A. (1967). Vapor pressures and triple point temperatures for several pure fluorocarbons. *Journal of Chemical and Engineering Data*, 12, 481–485.

[51] Mousat, A. H. N. (1978). Study of vapor pressure and critical properties of perfluoro-*n*-hexane. *Journal of Chemical and Engineering Data*, 23, 133–134.

[52] Dias, A. M. A., Gonçalves, C. M. B., Caço, A. I., Santos, L. M. N. B. F., Piñeiro, M. M., Vega, L. F., Coutinho, J. A. P., and Marrucho, I. M. (2005). Densities and vapor pressures of highly fluorinated compounds. *Journal of Chemical and Engineering Data*, 50, 1328–1333.

[53] Steele, W. V., Chirico, R. D., Knipmeyer, S. E., and Nguyen, A. (1997). Vapor pressure, heat capacity, and density along the saturation line, measurements for dimethyl isophthalate, dimethyl carbonate, 1,3,5-triethylbenzene, pentafluorophenol, 4-tert-butylcatechol, *a*-methylstyrene, and N,N'-bis(2-hydroxyethyl)ethylenediamine. *Journal of Chemical and Engineering Data*, 42, 1008–1020.

[54] Steele, W. V., Chirico, R. D., Knipmeyer, S. E., and Nguyen, A. (1997). Vapor pressure, heat capacity, and density along the saturation line, measurements for cyclohexanol, 2-cyclohexen-1-one, 1,2-dichloropropane, 1,4-di-tert-butylbenzene, (±)-2-ethylhexanoic acid, 2-(methylamino)ethanol, perfluoro-n-heptane, and sulfolane. *Journal of Chemical and Engineering Data*, 42, 1021–1036.

[55] Steele, W. V., Chirico, R. D., Knipmeyer, S. E., and Nguyen, A. (2002). Vapor pressure, heat capacity, and density along the saturation line: Measurements for benzenamine, butylbenzene, sec-butylbenzene, tert-butylbenzene, 2,2-dimethylbutanoic acid, tridecafluoroheptanoic acid, 2-butyl-2-ethyl-1,3-propanediol, 2,2,4-trimethyl-1,3-pentanediol, and 1-chloro-2-propanol. *Journal of Chemical and Engineering Data*, 47, 648–666.

[56] Steele, W. V., Chirico, R. D., Knipmeyer, S. E., and Nguyen, A. (2002). Measurements of vapor pressure, heat capacity, and density along the saturation line for cyclopropane carboxylic acid, N,N-diethylethanolamine, 2,3-dihydrofuran, 5-hexen-2-one, perfluorobutanoic acid, and 2-phenylpropionaldehyde. *Journal of Chemical and Engineering Data*, 47, 715–724.

[57] Kaiser, M. A., Larsen, B. S., Kao, C. P. C., and Buck, R. C. (2005). Vapor pressures of perfluorooctanoic, -nonanoic, -decanoic, -undecanoic, and -dodecanoic acids. *Journal of Chemical and Engineering Data*, 50, 1841–1843.

[58] Scott, L. S. (1986). Determination of activity coefficients by accurate measurement of boiling point diagram. *Fluid Phase Equilibria*, 26, 149–163.

[59] Washburn, S. T., Bingman, T. S., Braithwaite, S. K., Buck, R. C., Buxton, L. W., Clewell, H. J., Haroun, L. A., Kester, J. E., Rickard, R. W., and Shipp,

A. M. (2005). Exposure assessment and risk characterization for perfluorooctanoate in selected consumer articles. *Environmental Science and Technology*, 39, 3904–3910.

[60] Barton, C. A., Botelho, M. A., and Kaiser, M. A. (2008). Solid vapor pressure and enthalpy of sublimation for perfluorooctanoic acid. *Journal of Chemical and Engineering Data*, 53, 939–941.

[61] Barton, C. A., Botelho, M. A., and Kaiser, M. A. (2009). Solid vapor pressure and enthalpy of sublimation for ammonium perfluorooctanoate. *Journal of Chemical and Engineering Data*, 54, 752–755.

[62] Lei, Y. D., Wania, F., Mathers, D., and Mabury, S. A. (2004). Determination of vapor pressures, octanol-air, and water-air partition coefficients for polyfluorinated sulfonamide, sulfonamidoethanols, and telomer alcohols. *Journal of Chemical and Engineering Data*, 49, 1013–1022.

[63] Shoeib, M., Harner, T., Ikonomou, M., and Kannan, K. (2004). Indoor and outdoor air concentrations and phase partitioning of perfluoroalkyl sulfonamides and polybrominated diphenyl ethers. *Environmental Science and Technology*, 38, 1313–1320.

[64] Wania, F., Shiu, W.-Y., and Mackay, D. (1994). Measurement of the vapor pressure of several low-volatility organochlorine chemicals at low temperatures with a gas saturation method. *Journal of Chemical and Engineering Data*, 39, 572–577.

[65] Stock, N. L., Ellis, D. A., Deleebeeck, L., Muir, D. C. G., and Mabury, S. A. (2004). Vapor pressures of the fluorinated telomer alcohols: Limitations of estimation methods. *Environmental Science and Technology*, 38, 1693–1699.

[66] Krusic, P. J., Marchione, A. A., Davidson, F., Kaiser, M. A., Kao, C. C., Richardson, R. E., Botelho, M., Waterland, R. L., and Buck, R. C. (2005). Vapor pressure and intramolecular hydrogen bonding in fluorotelomer alcohols. *The Journal of Physical Chemistry A*, 109, 6232–6241.

[67] Goss, K., Bronner, G., Harner, T., Hertel, M., and Schmidt, T. C. (2006). The partition behavior of fluorotelomer alcohols and olefins. *Environmental Science and Technology*, 40, 3572–3577.

[68] Arp, H. P. H., Niederer, C., and Goss, K. (2006). Predicting the partitioning behavior of various highly fluorinated compounds. *Environmental Science and Technology*, 40, 7298–7304.

[69] Cobranchi, D. P., Botelho, M., Buxton, L. W., Buck, R. C., and Kaiser, M. A. (2006). Vapor pressure determinations of 8–2 fluorortelomer alcohol and 1-H perfluorooctane by capillary gas chromatography Relative retention time versus headspace methods. *The Journal of Chromatography A*, 1108, 248–251.

[70] Moroi, Y., Yano, H., Shibata, O., and Yonemitsu, T. (2001). Determination of acidity constants of perfluoroalkanoic acids. *Bulletin of the Chemical Society of Japan*, 74, 667–672.

[71] Burns, D. C., Ellis, D. A., Li, H., McMurdo, C. J., and Webster, E. (2008). Experimental p*K*a determination for perfluorooctanoic acid (PFOA) and the potential impact of pKa concentration dependence on laboratory-measured partitioning phenomena and environmental modeling. *Environmental Science and Technology*, 42, 9283–9288.

[72] McMurdo, C. J., Ellis, D. A., Webster, E., Butler, J., Christensen, R. D., and Reid, L. K. (2008). Aerosol enrichment of the surfactant PFO and mediation of the water-air transport of gaseous PFOA. *Environmental Science and Technology*, 42, 3969–3974.

[73] Goss, K. U. (2008). The p*K*a values of PFOA and other highly fluorinated carboxylic acids. *Environmental Science and Technology*, 42, 456–458.

[74] Goss, K. U. (2008). The p*K*a values of PFOA and other highly fluorinated carboxylic acids—correction. *Environmental Science and Technology*, 42, 5032–5032.

[75] Goss, K. U., and Arp, H. P. H. (2009). Comment on "Experimental pKa determination for perfluorooctanoic acid (PFOA) and the potential impact of pKa concentration dependence on laboratory-measured partitioning phenomena and environmental modeling". *Environmental Science and Technology*, 43, 5150–5151.

[76] Ellis, D. A., and Webster, E. (2009). Response to comment on "Aerosol enrichment of the surfactant PFO and mediation of the water-air transport of gaseous PFOA." *Environmental Science and Technology*, 43, 1234–1235.

[77] Rayne, S., Forest, K., and Friesen, K. J. (2009). Computational approaches may underestimate p*K*a values of longer-chain perfluorinated carboxylic acids: Implications for assessing environmental and biological effects. *Journal of Environmental Science and Health, Part A*, 44, 317–326.

[78] Rayne, S., Forest, K., and Friesen, K. (2009). Extending the semi-empirical PM6 method for carbon oxyacid p*K*a prediction to sulfonic acids: application toward congener-specific estimates for the environmentally and toxicologically relevant C1 through C8 perfluoroalkyl derivatives. *Nature Precedings*. Retrieved from http://hdl.handle.net/10101/npre.2009.2922.1

[79] Rayne, S., and Forest, K. (2009). Theoretical studies on the p*K*a values of perfluoroalkyl carboxylic acids: chain helicity. *Nature Precedings*. Retrieved from http://hdl.handle.net/10101/npre.2009.3829.1

[80] Rayne, S., and Forest, K. (2009). Theoretical studies on the p*K*a values of perfluoroalkyl carboxylic acids: Non-helical conformation acidity constants for n-perfluorooctanoic acid (n-PFOA). *Nature Precedings*. Retrieved from http://hdl.handle.net/10101/npre.2009.3837.2

[81] Brace, N. O. (1962). Long chain alkanoic and alkenoic acids with perfluoroalkyl terminal segments. *The Journal of Organic Chemistry*, 27, 4491–4498.

[82] Igarashi, S., and Yotsuyanagi, T. (1992). Homogeneous liquid-liquid extraction by pH dependent phase separation with a fluorocarbon ionic surfactant and its application to the preconcentration of porphyrin compounds. *Microchimica Acta*, 106, 37–44.

[83] López-Fontán, J. L., Sarmiento, F., and Schulz, P. C. (2005). The aggregation of sodium perfluorooctanoate in water. *Colloid and Polymer Science*, 283, 862–871.

[84] Barton, C. A., Kaiser, M. A., and Russell, M. H. (2007). Partitioning and removal of perfluorooctanoate during rain events: The importance of physical-chemical properties. *Journal of Environmental Monitoring*, 9, 839–846.

[85] Bowden, D. J., Clegg, S. L., and Brimblecombe, P. (1996). The Henry's law constant of trifluoroacetic acid and its partitioning into liquid water in the atmosphere. *Chemosphere*, 32, 405–420.

[86] Kutsuna, S., and Hori, H. (2008). Experimental determination of Henry's law constants of trifluoroacetic acid at 278–298 K. *Atmospheric Environment*, 42, 1399–1412.

[87] Li, H., Ellis, D., and Mackay, D. (2007). Measurement of low air-water partition coefficients of organic acids by evaporation from a water surface. *Journal of Chemical and Engineering Data*, 52, 1580–1584.

[88] Kah, M., and Brown, C. D. (2008). Log D: Lipophilicity for ionisable compounds. *Chemosphere*, 72, 1401–1408.

[89] Schwarzenbach, R. P., Gschwend, P. M., and Imboden, D. M. (2002). *Environmental organic chemistry* (2nd ed.). Hoboken, NJ: Wiley.

[90] Carmosini, N., and Lee, L. S. (2008). Partitioning of fluorotelomer alcohols to octanol and different sources of dissolved organic carbon. *Environmental Science and Technology*, 42, 6559–6565.

[91] Jing, P., Rodgers, P. J., and Amemiya, S. (2009). High lipophilicity of perfluoroalkyl carboxylate and sulfonate: Implications for their membrane permeability. *Journal of the American Chemical Society*, 131, 2290–2296.

[92] Thuens, S., Dreyer, A., Sturm, R., Temme, C., and Ebinghaus, R. (2008). Determination of the octanol-air partition coefficient (K_{OA}) of fluorotelomer alcohols. *Journal of Chemical and Engineering Data*, 53, 223–227.

[93] Dreyer, A., Langer, V., and Ebinghaus, R. (2009). Determination of octanol-air partition coefficients (*K*oa) of fluorotelomer acrylates, perfluoroalkyl sulfonamids, and perfluoroalkylsulfonamido ethanols. *Journal of Chemical and Engineering Data*, 54, 3022–3025.

[94] Higgins, C. P., and Luthy, R. G. (2006). Sorption of perfluorinated surfactants on sediments. *Environmental Science and Technology*, 40, 7251–7256.

[95] Mackay, D., and Fraser, A. (2000). Bioaccumulation of persistent organic chemicals: mechanisms and models. *Environmental Pollution*, 110, 375–391.

[96] Conder, J. M., Hoke, R. A., De Wolf, W., Russell, M. H., and Buck, R. C. (2008). Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds. *Environmental Science and Technology*, 42, 995–1003.

[97] Han, X., Snow, T. A., Kemper, R. A., and Jepson, G. W. (2003). Binding of perfluorooctanoic acid to rat and human plasma proteins. *Chemical Research in Toxicology*, 16, 775–781

[98] Biffinger, J. C., Kim, H. W., and DiMagno, S. G. (2004). The polar hydrophobicity of fluorinated compounds. *ChemBioChem*, 5, 622–627.

[99] Van de Vijver, K. I., Hoff, P., Das, K., Brasseur, S., Van Dongen, W., Esmans, E., Reijnders, P., Blust, R., and De Coen, W. (2005). Tissuedistribution of perfluorinated chemicals in harbor seals (*Phoca vitulina*) from the Dutch Wadden Sea. *Environmental Science and Technology*, 39, 6978–6984.

[100] Taniyasu, S., Kannan, K., Horii, Y., Hanari, N., and Yamashita, N. (2003). A survey of perfluorooctane sulfonate and related perfluorinated organic compounds in water, fish, birds and humans in Japan. *Environmental Science and Technology*, 37, 2634–2639.

[101] Ankley, G. T., Kuehl, D. W., Kahl, M. D., Jensen, K. M., Butterworth, B. C., and Nichols, J. W. (2004). Partial life-cycle toxicity and bioconcentration modeling of perfluorooctanesulfonate in the northern leopard frog (*rana pipiens*). *Environmental Toxicology and Chemistry*, 23, 2745–2755.

[102] Kannan, K., Tao, L., Sinclair, E., Pastva, S. D., Jude, D. J., and Giesy, J. P. (2005). Perfluorinated compounds in aquatic organisms at various trophic levels in a Great Lakes food chain. *Archives of Environmental Contamination and Toxicology*, 48, 559–566.

[103] Sinclair, E., Mayack, D. T., Roblee, K., Yamashita, N., and Kannan, K. (2006). Occurrence of perfluoroalkyl surfactants in water, fish, and birds from New York State. *Archives of Environmental Contamination and Toxicology*, 50, 398–410.

[104] Morikawa, A., Kamei, N., Harada, K., Inoue, K., Yoshinaga, T., Saito, N., and Koizumi, A. (2006). The bioconcentration factor of perfluorooctane sulfonate is significantly larger than that of perfluorooctanoate in wild turtles (*Trachemys scripta elegans* and *Chinemys reevessii*): An Ai River ecological study in Japan. *Ecotoxicology and Environmental Safety*, 65, 14–21.

[105] Houde, M., Bujas, T. A. D., Small, J., Wells, R. S., Fair, P. A., Bossart, G. D., Solomon, K. R., and Muir, D. C. G. (2006). Biomagnification of perfluoroalkyl compounds in the bottlenose dolphin (*Tursiops truncatus*) food web. *Environmental Science and Technology*, 40, 4138–4144.

[106] Furdui, V., Stock, N., Ellis, D. A., Butt, C. M., Whittle, M., Crozier, P. W., Reiner, E. J., Muir, D. C. G., and Mabury, S. (2007). Spatial distribution of perfluoroalkyl contaminants in lake trout from the Great Lakes. *Environmental Science and Technology*, 41, 1554–1559.

[107] Haukås, M., Berger, U., Hop, H., Gulliksen, B., and Gabrielsen, G. W. (2007). Bioaccumulation of polyfluorinated alkyl substances (PFAS) in selected species from the Barents Sea food web. *Environmental Pollution*, 148, 360–371.

[108] Higgins, C. P., McLeod, P. B., MacManus-Spencer, L. A., and Luthy, R. G. (2007). Bioaccumulation of perfluorochemicals in sediments by the aquatic oligochaete *Lumbriculus variegatus*. *Environmental Science and Technology*, 41, 4600–4606.

[109] Houde, M., Czub, G., Small, J. M., Backus, S., Wang, X. W., Alaee, M., and Muir, D. C. G. (2008). Fractionation and bioaccumulation of perfluorooctane sulfonate (PFOS) isomers in a Lake Ontario food web. *Environmental Science and Technology*, 42, 9397–9403.

[110] Boudreau, T. M., Sibley, P. K., Mabury, S. A., Muir, D. C. G., and Solomon, K. R. (2003). Laboratory evaluation of the toxicity of perfluorooctane sulfonate (PFOS) on *Selenastrum capricornutum*, *Chlorella vulgaris*, *Lemna gibba*, *Daphnia magna*, and *Daphnia pulicaria*. *Archives of Environmental Contamination and Toxicology*, 44, 307–313.

[111] Hanson, M. L., Sibley, P. K., Brain, R. A., Mabury, S. A., and Solomon, K. R. (2005). Microcosm evaluation of the toxicity and risk to aquatic macrophytes from perfluorooctane sulfonic acid. *Archives of Environmental Contamination and Toxicology*, 48, 329–337.

[112] Hanson, M. L., Small, J., Sibley, P. K., Boudreau, T., Brain, R. A., Mabury, S. A., and Solomon, K. R. (2005). Microcosm evaluation of the fate, toxicity and risk to aquatic macrophytes from perfluorooctanoic acid (PFOA). *Archives of Environmental Contamination and Toxicology*, 49, 307–316.

[113] Phillips, M. M., Dinglasan-Panlilio, M. J., Mabury, S. A., Solomon, K. R., and Sibley, P. K. (2007). Fluorotelomer acids are more toxic than perfluorinated acids. *Environmental Science and Technology*, 41, 7159–7163.

[114] Li, M. H. (2009). Toxicity of perfluorooctane sulfonate and perfluorooctanoic acid to plants and aquatic invertebrates. *Environmental Toxicology*, 24, 95–101.

[115] Colombo, I., de Wolf, W., Thompson, R. S., Farray, D. G., Hoke, R. A., and L'Haridon, J. L. (2008). Acute and chronic aquatic toxicity of ammonium perfluorooctanoate (APFO) to freshwater organisms. *Ecotoxicology and Environmental Safety*, 71, 749–756.

[116] Liu, W., Chen, S., Quan, X., and Jin, Y. H. (2008). Toxic effect of serial perfluorosulfonic and perfluorocarboxylic acids on the membrane system of a freshwater alga measured by flow cytometry. *Environmental Toxicology and Chemistry*, 27, 1597–1604.

[117] Liu, W., Zhang, Y. B., Quan, X., Jin, Y. H., and Chen, S. (2009). Effect of perfluorooctane sulfonate on toxicity and cell uptake of other compounds with different hydrophobicity in green alga. *Chemosphere*, 75, 405–409.

[118] Latała, A., Nędzi, M., and Stepnowski, P. (2009). Acute toxicity assessment of perfluorinated carboxylic acids toward the Baltic microalgae. *Environmental Toxicology and Pharmacology*, 28, 167–171.

[119] Li, M. H. (2010). Chronic effects of perfluorooctane sulfonate and ammonium perfluorooctanoate on biochemical parameters, survival and reproduction of *Daphnia magna*. *Journal of Health Science*, 56, 104–111.

[120] Ji, K., Kim, Y., Oh, S., Ahn, B., Jo, H., and Choi, K. (2008). Toxicity of perfluorooctane sulfonic acid and perfluorooctanoic acid on freshwater macroinvertebrates (*Daphnia magna* and *Moina macrocopa*) and fish (*Oryzias latipes*). *Environmental Toxicology and Chemistry*, 27, 2159–2168.

[121] Phillips, M. M., Dinglasan-Panlilio, M. J., Mabury, S. A., Solomon, K. R., and Sibley, P. K. (2010). Chronic toxicity of fluorotelomer acids to *Daphnia magna* and *Chironomus dilutus*. *Environmental Toxicology and Chemistry*, 29, 1123–1131.

[122] MacDonald, M. M., Warne, A. L., Stock, N. L., Mabury, S. A., Solomon, K. R., and Sibley, P. K. (2004). Toxicity of perfluorooctane sulfonic acid and perfluorooctanoic acid to *Chironomus tentans*. *Environmental Toxicology and Chemistry*, 23, 2116–2123.

[123] Wang, Z. Y., Ud-Daula, A, Fiedler, S., and Schramm, K. W. (2010). Impact of fluorotelomer alcohols (FTOH) on the molecular and macroscopic phenotype of *Tetrahymena thermophila*. *Environmental Science and Pollution Research International*, 17, 154–164.

[124] Sanderson, H., Boudreau, T. M., Mabury, S. A., Cheong, W. J., and Solomon, K. R. (2002). Ecological impact and environmental fate of perfluorooctane sulfonate on the zooplankton community in indoor microcosms. *Environmental Toxicology and Chemistry*, 21, 1490–1496.

[125] Sanderson, H., Boudreau, T. M., Mabury, S. A., and Solomon, K. R. (2003). Impact of perfluorooctanoic acid on the structure of the zooplankton community in indoor microcosms. *Aquatic Toxicology*, 62, 227–234.

[126] Sanderson, H., Boudreau, T. M., Mabury, S. A., and Solomon, K. R. (2004). Effects of perfluorooctane sulfonate and perfluorooctanoic acid on the zooplanktonic community. *Ecotoxicology and Environmental Safety*, 58, 68–76.

[127] Mulkiewicz, E., Jastorff, B., Skałdanowski, A. C., Kleszczyński, K., and Stepnowski, P. (2007). Evaluation of the acute toxicity of perfluorinated carboxylic acids using eukaryotic cell lines, bacteria and enzymatic assays. *Environ Toxicol Pharmacol*, 23, 279–285.

[128] Ankley, G. T., Kuehl, D. W., Kahl, M. D., Jensen, K. M., Linnum, A., Leino, R. L., and Villeneuvet, D. A. (2005). Reproductive and developmental toxicity and bioconcentration of perfluorooctanesulfonate in a partial life-cycle test with the fathead minnow (*Pimephales promelas*). *Environmental Toxicology and Chemistry*, 24, 2316–2324.

[129] Huang, H. H., Huang, C. J., Wang, L. J., Ye, X. W., Bai, C. L., Simonich, M. T., Tanguay, R. L., and Dong, Q. X. (2010). Toxicity, uptake kinetics and behavior assessment in zebrafish embryos following exposure to perfluorooctanesulphonicacid (PFOS). *Aquatic Toxicology*, 98, 139–147.

[130] Hoff, P. T., Van Dongen, W., Esmans, E. L., Blust, R., and De Coen, W. M. (2003). Evaluation of the toxicological effects of perfluorooctane sulfonic acid in the common carp (*Cyprinus carpio*). *Aquatic Toxicology*, 62, 349–359.

[131] Oakes, K., Sibley, P. K., Martin, J. M., Maclean, D. D., Solomon, K. R., Mabury, S. A., and Van der Kraak, G. (2005). Short-term exposures of fish to perfluorooctane sulfonate: acute effects on fatty acyl-CoA oxidase activity, oxidative stress, and circulating sex steroids. *Environmental Toxicology and Chemistry*, 24, 1172–1181.

[132] Hagenaars, A., Knapen, D., Meyer, I. J., van der Ven, K., Hoff, P., and De Coen, W. (2008). Toxicity evaluation of perfluorooctane sulfonate (PFOS) in the liver of common carp (*Cyprinus carpio*). *Aquatic Toxicology*, 88, 155–163.

[133] Wei, Y. H., Dai, J. Y., Liu, M., Wang, J. S., Xu, M. Q., Zha, J. M., and Wang, Z. J. (2007). Estrogenlike properties of perfluorooctanoic acid as revealed by expressing hepatic estrogen-responsive genes in rare minnows (*Gobiocypris rarus*). *Environmental Toxicology and Chemistry*, 26, 2440–2447.

[134] Liu, Y., Wang, J. S., Wei, Y. H., Zhang, H. X., Liu, Y., and Dai, J. Y. (2008). Molecular characterization of cytochrome P450 1A and 3A and the effects of perfluorooctanoic acid on their mRNA levels in rare minnow (*Gobiocypris rarus*) gills. *Aquatic Toxicology*, 88, 183–190.

[135] Liu, Y., Wang, J. S., Liu, Y., Zhang, H. X., Xu, M. Q., and Dai, J. Y. (2009). Expression of a novel cytochrome P450 4T gene in rare minnow (*Gobiocypris rarus*) following perfluorooctanoic acid exposure. *Comparative Biochemistry and Physiology, Part C*, 150, 57–64.

[136] Liu, Y., Wang, J. S., Wei, Y. H., Zhang, H. X., Xu, M. Q., and Dai, J. Y. (2008). Induction of time-dependent oxidative stress and related transcriptional effects of perfluorododecanoic acid in zebrafish liver. *Aquatic Toxicology*, 89, 242–250.

[137] Wei, Y. H., Chan, L. L., Wang, D. Z., Zhang, H. X., Wang, J. S., and Dai, J. Y. (2008). Proteomic analysis of hepatic protein profiles in rare minnow

(*Gobiocypris rarus*) exposed to perfluorooctanoic acid. *Journal of Proteome Research*, 7, 1729–1739.

[138] Wei, Y. H., Liu, Y., Wang, J. S., Tao, Y., and Dai, J. Y. (2008). Toxicogenomic analysis of the hepatic effects of perfluorooctanoic acid on rare minnows (*Gobiocypris rarus*). *Toxicology and Applied Pharmacology*, 226, 285–297.

[139] Wei, Y. H., Shi, X. J., Zhang, H. X., Wang, J. S., Zhou, B. S., and Dai, J. Y. (2009). Combined effects of polyfluorinated and perfluorinated compounds on primary cultured hepatocytes from rare minnow (*Gobiocypris rarus*) using toxicogenomic analysis. *Aquatic Toxicology*, 95, 27–36.

[140] Liu, C. S., Yu, K., Shi, X. J., Wang, J. X., Lam, P. K.mS., Wu, R. S. S., and Zhou, B. S. (2007). Induction of oxidative stress and apoptosis by PFOS and PFOA in primary cultured hepatocytes of freshwater tilapia (*Oreochromis niloticus*). *Aquatic Toxicology*, 82, 135–143.

[141] Liu, C. S., Du, Y. B., and Zhou, B. S. (2007). Evaluation of estrogenic activities and mechanism of action of perfluorinated chemicals determined by vitellogenin induction in primary cultured tilapia hepatocytes. *Aquatic Toxicology*, 85, 267–277.

[142] Liu, C. S., Yu, L. Q., Deng, J., Lam, P. K. S., Wu, R. S. S., and Zhou, B. S. (2009). Waterborne exposure to fluorotelomer alcohol 6:2 FTOH alters plasma sex hormone and gene transcription in the hypothalamic–pituitary–gonadal (HPG) axis of zebrafish. *Aquatic Toxicology*, 93, 131–137.

[143] Liu, C. S., Deng, J., Yu, L. Q., Ramesh, M., and Zhou, B. S. (2010). Endocrine disruption and reproductive impairment in zebrafish by exposure to 8:2 fluorotelomer alcohol. *Aquatic Toxicology*, 96, 70–76.

[144] Shi, X. J., Du, Y. B., Lam, P. K. S., Wu, R. S. S., and Zhou, B. S. (2008). Developmental toxicity and alteration of gene expression in zebrafish embryos exposed to PFOS. *Toxicology and Applied Pharmacology*, 230, 23–32.

[145] Shi, X. J., Yeung, L. W. Y., Lam, P. K. S., Wu, R. S. S., and Zhou, B. S. (2009). Protein profiles in zebrafish (*Danio rerio*) embryos exposed to perfluorooctane sulfonate. *Toxicological Sciences*, 110, 334–340.

[146] Shi, X. J., Liu, C. S., Wu, G. Q., and Zhou, B. S. (2009). Waterborne exposure to PFOS causes disruption of the hypothalamus–pituitary–thyroid axis in zebrafish larvae. *Chemosphere*, 77, 1010–1018.

[147] Du, Y. B., Shi, X. J., Liu, C. S., Yu, K., and Zhou, B. S. (2009). Chronic effects of water-borne PFOS exposure on growth, survival and hepatotoxicity in zebrafish: A partial life-cycle test. *Chemosphere*, 74, 723–729.

[148] Fang, X. M., Wei, Y. H., Liu, Y., Wang, J. S., and Dai, J. Y. (2010). The identification of apolipoprotein genes in rare minnow (*Gobiocypris rarus*) and their expression following perfluorooctanoic acid exposure. *Comparative Biochemistry and Physiology, Part C*, 151, 152–159.